ASPECTS OF TACTICAL BIOLOGICAL DEFENSE

A thesis presented to the Faculty of the U.S. Army Command and General Staff College in partial fulfillment of the requirements for the degree

MASTER OF MILITARY ART AND SCIENCE

by

TIMOTHY F. MOSHIER, MAJOR, USA
B.A., State University of New York College at Oswego, New York, 1981
M.S., Syracuse University, Syracuse, New York, 1990

Fort Leavenworth, Kansas
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Aspects of Tactical Biological Defense

Major Timothy F. Moshier, USA

U.S. Army Command and General Staff College
ATTN: ATZL-SWD-GD
Ft. Leavenworth, Kansas 66027-6900

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The threat of biological warfare (BW) directed against our forces is greater today than at any other time in the history of modern warfare. This thesis represents the first attempt to answer the question "What is an effective design for tactical biological defense?" Established criteria for agents of biological origin (ABOs) are analyzed for their applicability to tactical operations. Potential ABOs are evaluated for their usefulness on the tactical battlefield. Information requirements (IRs) for use in intelligence preparation of the battlefield (IPB) are developed. Known and potential delivery means are listed. Analysis of the respiratory threat is made. Mathematical modelling of potential biological attack scenarios is used to determine BW's potential for limiting forces' freedom of action, and for developing detection requirements and vulnerability assessment tools. Candidate detection technologies are reviewed, and a battlefield detection strategy is developed. Finally, critical tasks for biological detection units are formulated.

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THESIS APPROVAL PAGE

Name of Candidate: Major Timothy F. Moshier

Thesis Title: Aspects of Tactical Biological Defense

Approved by:

_____, Thesis Committee Chairman
Major Henry J. Davis, B.S.

_____, Member
Major Geoffrey L. Gresham, M.A.

_____, Member
Colonel Jerry W. Warner, Ph.D.

Accepted this 3rd day of June 1994 by:

_____, Director, Graduate Degree Programs
Philip J. Brookes, Ph.D.

The opinions and conclusions expressed herein are those of the student author and do not necessarily represent the views of the U.S. Army Command and General Staff College or any other governmental agency. (References to this study should include the foregoing statement.)

ABSTRACT

ASPECTS OF TACTICAL BIOLOGICAL DEFENSE by MAJ Timothy F. Moshier, USA, 146 pages.

The threat of biological warfare (BW) directed against our forces is greater today than at any other time in the history of modern warfare. This thesis represents the first attempt to answer the question "What is an effective design for tactical biological defense?" Established criteria for agents of biological origin (ABOs) are analyzed for their applicability to tactical operations. Potential ABOs are evaluated for their usefulness on the tactical battlefield. Information requirements (IRs) for use in intelligence preparation of the battlefield (IPB) are developed. Known and potential delivery means are listed. Analysis of the respiratory threat is made. Mathematical modelling of potential biological attack scenarios is used to determine BW's potential for limiting forces' freedom of action, and for developing detection requirements and vulnerability assessment tools. Candidate detection technologies are reviewed, and a battlefield detection strategy is developed. Finally, critical tasks for biological detection units are formulated.

ACKNOWLEDGEMENTS

I had two goals in writing this thesis. My first goal was to answer my own questions--what is BW all about, and what do we have to do to defeat the BW threat on the battlefield? My second goal was to provide soldiers a "tool box" of techniques and analyses that will assist them in understanding a very difficult issue, and will prepare them to meet a very real contingency.

In meeting both of these goals, I have received invaluable guidance from my thesis committee members: MAJ Joe Davis and MAJ Geoffrey Greetham of the Center for Army Tactics, and COL Jerry Warner of Northern Kentucky University. At a time when everyone is expected to do ever more with ever less, these three were able to dedicate some of their scarce time to assist me. And I sincerely appreciate that.

Of course, I must also acknowledge the absolutely necessary support I have received on the home front. To that end, I thank my wife Mary Ann, who has demonstrated untiring patience (again), and my four-and-one-half year old daughter Susan -- for not playing with the computer.

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INTRODUCTION

Why A Concern With Biological Defense

> We won't copy you anymore, making planes to catch up with your planes, missiles to catch up with your missiles. We'll take asymmetrical means with new scientific principles available to us. Genetic engineering could be a hypothetical example. Things can be done for which neither side could find defenses or countermeasures. . . These are not just words. I know what I'm saying. (Valentin Falin, Soviet Novosti Press Agency, 1987)[1]

Biological weapons are weapons of mass destruction, and the potential for their employment against our forces is greater today than at any other time in the history of modern warfare. Society's abhorrence of biological weapons is well documented, and governments have been pursuing effective deterrents against their use since the early part of this century. Examples of attempts to discourage use of these weapons include the Geneva Protocol of 1925, and the Biological Weapons Convention of 1972.[2] Unfortunately, the existing threat proves that diplomatic efforts alone are insufficient to protect our forces from the effects of biological warfare. Adequate force protection can only be attained through the establishment of an effective biological defense program.

Background

Biological warfare (BW) has been a specter floating at the fringes of our defense concerns since the end of the first world war, but instead of fading into the realm of obsolescence it has recently emerged as a rapidly growing threat to our forces. In fact, the number of nations that are known to possess the capability and interest to make offensive use of biological agents has grown in the past thirteen years from

three to ten, and another four nations are suspected of having offensive biological warfare programs.[3] The predominant arguments for dismissal of the biological threat had been the fact that microbes (i.e., bacteria, viruses, rickettsia and other disease causing organisms) are difficult to control once released into the environment, and that biological agents usually have a significant lag time before manifestation of effects (e.g., days to weeks). These arguments are most valid when the threat is restricted to the use of microbes, and when the anticipated scenario for conflict is against a well armed opponent with a conventional arsenal equivalent to or greater than our own. But now the threat has changed. The life sciences have been revolutionized in the past thirty years, and our opponents are likely to be struggling autocracies ready to exploit any form of combat power available to them.

Biological warfare, for all its social disapprobation, does have several characteristics which makes it appealing to some of the belligerents we are likely to face within the next 20 years. These characteristics include: a wide range of agents, specificity of action, availability of vaccines, economy, deniability of employment, and the inherent power that comes with being the possessor of weapons of mass destruction.

Biological agents are no longer limited to the "bacteriological" class of agents which was specified in the 1925 Geneva Protocol. A biological arsenal may include a wide variety of naturally occurring toxins[4] and physiological regulators, which may have similar employment characteristics to chemical agents. Also, the employer of biological agents may be able to immunize his forces against the agent, and thus operate with minimum degradation on the contaminated battlefield, while the target force is required to wear cumbersome protective gear.

Another reason for belligerents to use biological agents is their economy. A group of experts testified to a United Nations panel in 1969 that "for large-scale

operation against a civilian population, casualties might cost about $2,000 per square kilometer with conventional weapons, $800 with nuclear weapons, $600 with nerve-gas weapons, and $1 with biological weapons."[5] Even with corrections for inflation one can see the economic benefits of using biological agents. Since this testament was given to the United Nations, advancements in genetic engineering and chemical synthesis[6] have made production of formerly rare agents (e.g., many biological toxins) more affordable.

One of the dangers that a belligerent faces in using weapons of mass destruction is the possibility of severe retribution from the international community; perhaps even the reciprocal use of weapons of mass destruction. But the detection of some types of biological attack are extremely difficult. Consider the difficulties that might be associated with differentiating between an outbreak of cholera caused by an offensive employment of the causative agent *Vibrio cholerae*, and a natural outbreak of the organism, which has already caused seven epidemics.[7] The superficial similarities between the two cases underscores the need for a sophisticated BW detection and identification strategy. A strategy that will alert our forces to take the proper protective measures only when pathogens reach tactically significant concentrations, and not during natural fluctuations. Avoiding degradation due to unnecessary wear of protective gear is just as important as avoiding degradation due to BW attack.

The trepidation that afflicts forces aware of an opponent's BW capability will encourage them to don protective gear unnecessarily, which in turn will degrade their combat effectiveness. Thus, possession of biological weapons can lend a certain flexibility and defence to a belligerent. The threat of biological weapons employment must always be a concern to neighboring states with whom the possessor of biological weapons has a quarrel. Also, a state may develop and produce biological weapons as a deterrent to aggression against the state by its enemies. In the end, biological weapons

may be used both as deterrent (àla the cold war nuclear deterrent), and as battlefield combat multiplier.

Whatever the reason(s) for our potential enemys' possession of offensive biological capabilities, the US must have a credible deterrence program. In addition to diplomatic pressure, effective deterrence measures must include: conclusive, real-time detection and identification of biological attacks; chemo- and immuno-prophylactic measures; effective medical treatment protocols; operational protective measures; and appropriate retaliatory responses. Brief study of each of the deterrence measures reveals that they are interrelated to each other. Before appropriate prophylaxes can be taken, a valid operational assessment of the threat must be made. The occurrence of high concentrations of biological agents must be unquestionably proven to be due to an offensive act (and not simply a natural fluctuation in the background concentration) before alarming forces and having them expend the time and resources to take heightened protective measures. If, in fact, a biological attack is confirmed, then treatments will be required at significant logistical costs to the defender. Before appropriate retaliatory measures can be taken, there must be conclusive evidence of the belligerent's illegal use of biological weapons. When our armed forces are involved, the level at which all these protective measures (i.e., deterrence measures) must come together is the tactical level.

The Research Question

The preceding section enumerates the reasons why we must take the biological threat seriously. The next step is to ask what must be done to counter the threat in the form of a researchable question. The question that I will attempt to answer in this study is "What is an effective design for tactical biological defense?" This single question, however, is too broad to be handled effectively, and so I will approach it through a set of subordinate research questions. The subordinate research questions

are framed in a manner similar to the mission analysis sequence that is used to evaluate an area of operations/area of interest, and unit mission. I have chosen this framework partly because of its proven utility in military planning, but predominantly because my goal in writing this thesis is to provide the chemical defense officer with a useable kit of operational planning tools.

Subordinate Research Questions

What Are The Intelligence Requirements (IRs) For Biological Defense?

The chemical staff officer who is tasked with planning for nuclear, biological, and chemical (NBC) defense in a proposed theater of operations will have a number of questions that he will be asking to determine what, if any, capabilities the enemy has to conduct offensive NBC operations. But because the range of biological weapons is so broad, their employment so flexible, and our current state of training of chemical officers in biological defense so lacking, there is a need to develop a "standard kit" of biological defense intelligence requirements (IRs). This study will review the current biological threats and deduce a set of IRs which can be applied to a variety of theaters of operations.

What Conditions Support Employment Of Biological Weapons?

Many of the same conditions that support effective employment of chemical agents also support employment of biological agents. But there are additional considerations that a chemical officer must consider in the analysis of his unit's vulnerability to biological attack. Environmental, topographic, and tactical conditions must be considered with reference to the enemy's tactical objectives. Chemical Defence Officers must consider the pace of operations. If operations are at a standstill, and no significant actions are foreseen, then the enemy may take advantage of this condition to employ biological agents which may take several days to manifest their effects, but

possess other favorable characteristics. This question explores the validity and necessity of these considerations.

What Is Required For Conclusive Identification Of A Biological Attack?

The difficulty in identifying a biological attack may best be illustrated through a simple comparison. Nerve agent sarin (GB) does not occur naturally, and so any detection of it identifies a chemical attack. On the other hand, *Coxiella burnetii*, the causative agent of Q-fever, is endemic to many areas of the world and detection of it may simply reflect the natural background concentrations of the organism. This question examines the characteristics of offensively employed biological agents, potential detection strategies, and what is required to confirm an actual biological attack.

What Are The Downwind Hazard Profiles Of Several Biological Attacks?

To investigate and answer this question I have developed several biological attack scenarios, and modelled them to illustrate their downwind hazard. These models are not the same as the generic models that are described in Army Field Manual 3-3 Chemical And Biological Contamination Avoidance.[8] The modelling technique that is used in this study takes into consideration atmospheric stability, pathogen/toxin decay rates, respiration factors, and terrain characteristics.

Can Biological Attacks Affect Tactical "Centers Of Gravity?"

Center of gravity is defined by the Army as "that characteristic, capability, or location from which enemy and friendly forces derive their freedom of action, physical strength, or will to fight."[9] At the tactical level, centers of gravity may include reserves, logistic support bases, and command centers. Attack scenarios and downwind hazard models are used to examine this question. Products from this portion

of the study are useful to the chemical defense officer in risk analysis, and in planning and implementing effective defensive measures.

What Are The Critical Tasks For The Battlefield Biological Detection And Identification Unit?

Elliot A. Cohen and John Gooch argued that before a commander can successfully employ his unit he must understand the threat, the operational situation, unit weaknesses in light of the threat, and the unit's critical tasks.[10] In view of this reasoning, the biological detection units that are scheduled for activation around fiscal year 1996--1997 must have a list of critical tasks available to them to facilitate their training and operational doctrine development. While it would be presumptuous to believe that this study has identified all the critical tasks, the models and analyses from this study have helped to identify at least some of the tasks, and provide a basis for development of others.

Scope Of This Research

The scope of this research is limited to an analysis of tactical-level biological defense. I have used only unclassified sources for threat analyses and modelling data so that this document may receive widest dissemination. While immunization programs and post-attack medical treatment are important aspects of a total biological protection program, I have addressed them only in terms of how their availability may affect operaional decisions. Finally, I did not attempt to identify nations that possess BW programs in this thesis--classified intelligence sources can provide better data on this point than I can.

Assumptions

Assumptions I am making in support of this research include:

1. The mass spectrometer which is currently a part of the M93 FOX Nuclear, Biological, Chemical (NBC) Reconnaissance System may be programmed for the detection and identification of certain solvents and low molecular weight biochemicals.

2. Biological detection units will have access to medical (i.e., pathology) laboratory support within the theater of operations.

3. Biological vectors such as rats, mosquitos and ticks will not be considered at the tactical level because of the difficulty in preparing them, and the lack of control once they're released on the battlefield.

Endnotes

[1]Stephen Rose, "The Coming Explosion Of Silent Weapons," Naval War College Review 42 (Summer 1989), 10 - 11.

[2]Barend ter Haar, The Future Of Biological Weapons (New York: Praeger, 1991), 1 to 17.

[3]U.S. Army Chemical School, Unclassified Nuclear, Biological, Chemical Threat Briefing, 4 August 1993.

[4]Departments of the Army, Navy and Air Force, FM3-9, NAVFAC P-467, AFR 355-7, Potential Military Chemical/Biological Agents and Compounds, (Washington, D.C.: Government Printing Office, December 1990), 72 - 90.

[5]Neil C. Livingstone, and Joseph C. Douglass, Jr., CBW: The Poor Man's Atomic Bomb, (Cambridge, Massachusetts: Institute for Foreign Policy Analysis, Inc., 1984), 7.

[6]David Bradley, "Frog Venom Cocktail Yields a One-Handed Painkiller," Science 261 (27 August 1993), 1117.

[7]Richard Stone, "New Cholera Strain Arouses Concern," Science 261 (27 August 1993), 1119.

[8]U.S. Army and Marine Corps, FM 3-3/FMFM 11-17, Chemical And Biological Contamination Avoidance, (Washington, D.C.: Department of the Army, 1992), 4-4 to 4-20.

[9]U.S. Army, FM 100-5, Operations, (Washington, D.C.: Government Printing Office, 14 June 1993), 6-7.

[10]Elliot A. Cohen, and John Gooch, "What Can Be Done?" Military Misfortunes: The Anatomy Of Failure In War, (The Free Press, a division of Macmillan, Inc., 1990), 231 to 246; excerpt reprinted in U.S. Army Command and General Staff College, C610 Term I Syllabus/Book Of Readings (Fort Leavenworth: USACGSC, August 1993), 96 to 109.

CHAPTER 2

A HISTORICAL PERSPECTIVE

A historical review of biological warfare provides useful tools for evaluating today's biological threat. History offers insights into why belligerents chose to use biological warfare (BW), and what conditions support use of BW. One thing that becomes apparent from studying BW's history is the fact that it has been used so consistently. But even more remarkable are the ostensible repetitions of the patterns that supported past use of BW occurring today. In this chapter the author reviews the history of BW in reference to contemporary technological, socio-political and battlefield dynamics, and derives a set of conditions that indicate a high threat of BW.

Before 1914

Toxins and poisons have been a part of belligerents' arsenals since ancient times. As early as 600 BC the Greeks employed a strong diarrhetic derived from the roots of the heleborus plant to poison their enemy's drinking water.[1] In 200 BC the Carthaginians employed the narcotic mandragora root in a clever deception. Mixed in stocks of wine left in an abandoned position, the toxin debilitated the Carthaginians' enemies, and secured the their victory.[2]

From at least the early twelfth century to the middle nineteenth century warring parties introduced diseases to their enemies' camps by contaminating water sources with infected carcasses. Notable uses of this tactic include: the German Emperor Frederick Barbarossa's poisoning of water supplies in the Italian town of

Tortuna in 1155;[3] the Mongols' catapulting of plague-infected cadavers over the walls of the Crimean seaport city of Caffa in 1346 (which some hypothesize caused the infamous European "Black Death" epidemic);[4] and General Johnston, of the American Confederate States, who poisoned water sources around Vicksburg with dead pigs and sheep in July 1863 to slow the Union Army's advance.[5]

Belligerents of this period developed some especially novel delivery means to spread diseases. A sixteenth century Italian tactical manual "described how to construct artillery shells for delivery of disease to the enemy."[6] Although there are no known accounts of this invention actually being used, there is at least one record of an entrepreneurial Italian chemist who tried to sell it to Louis XIV.[7] In 1763 the British Commander-In-Chief Sir Jeffery Amherst suggested a method for defeating aggressive North American Indians. Sir Amherst's suggestion was to "send the *Small Pox* [sic] among those disaffected tribes...," and later recommended using inoculated blankets from British small pox hospitals as a means for spreading the disease.[8] Apparently, it was a Captain Ecuyer who, in June 1763, affected the transfer of two infected blankets and one handkerchief to two Ohio Indian Chiefs. This quiet coup triggered a devastating epidemic within several Indian tribes.[9]

There are several important observations to be made about biological warfare during this period in history. First, incapacitating agents may be significant combat multipliers--the chosen biological agent does not have to be lethal. Second, biological agents lend themselves to a wide variety of delivery means. And third, biological agents were most often used by a belligerent when conventional means alone were insufficient to overcome his adversary (e.g., the Mongol siege of Caffa and Johnston's poisoning of water sources as he retreated in front of the stronger Union Army).

Robin Clarke argued that biological weapons were not employed more extensively because belligerents didn't fully understand BW's technical aspects.[10] But it was during the latter half of the nineteenth century and the early part of the twentieth century that Robert Koch performed his pioneering work in microbiology, and established the causal relationships between certain microbial organisms and diseases.[11] For the first time, etiologic agents could be isolated from an infected host, selectively grown in the laboratory, and reintroduced into new hosts to repeat the disease. Now a degree of predictability and control of biological agents was possible. The biological sciences were about to become part of belligerents' arsenals.

1914 to 1945

It was during this period of history, covering the span of two world wars, that the sciences and the demands of total war combined to effect remarkable advances in the use of biological weapons.

The first allegations of biological warfare were made by the Allies against Germany during World War I. The Allies claimed in 1915, 1916 and 1917 that livestock had been inoculated by German agents with anthrax and glanders.[12] These alleged events, had they been confirmed, would have been the first biological attacks in which specific micro-organisms were selected and employed in pure form. Regardless whether these attacks were factual or not, BW was to make significant strides in the next thirty years.

Several significant milestones occurred during the inter-war and World War II years that lent unusual impetus to the development of biological warfare. It was during this time that the international community began to weigh the impact of the new sciences on warfare, and that national powers, with their very survival threatened, began to explore all possible advantages. Perhaps the earliest indicator of the role that

the various sciences were to play in World War II came from Fritz Haber in 1919. At his award ceremony for the Nobel Prize for Chemistry, Haber is cited as saying, "In no future war will the military be able to ignore poison gas. It is a higher form of killing."[13] Apparently, this was not an isolated attitude. The following quotation, which is from 1933, has been attributed to another German scientist:

> [biological warfare] is undoubtedly the given weapon for a nation that has been disarmed and is defenseless. . . It cannot be taken ill of such a nation if one day it defends itself by this means against brutal violation and destroys its oppressors by purely scientific means. . .[W]hen the existence of a state and nation is at stake every method is permissible to stave off the superior enemy and to vanquish him.[14]

Scientists' motivation for making these statements may have been a product more of professional conceit than of military or humanitarian reasoning. But their impact was not lost on at least one other scientist.

Shiro Ishii was an ambitious Japanese nobleman and physician who joined the Imperial Japanese Army in 1922[15] and went on to command one of the world's most aggressive biological warfare programs. The open, and apparently accepted, use of chemical warfare in Europe during World War I did not go unnoticed by Ishii. Chemical warfare's arguable unlawfulness under the Hague Convention[16] made its use especially impressive, and implied that other unconventional forms of warfare were acceptable. Ishii's intense interest in diseases' destructive potential,[17] combined with his ability to convince his superiors that BW was not only acceptable but already a part of their enemies' arsenals[18] made it possible for him to convince his superiors of BW's utility to the nation's cause--and to turn Japan into a major BW power.

Study of this period yields several important pieces of information. First, serious consideration for BW increases when scientific advances obviate the technical hurdles that would otherwise prevent offensive employment of biologics. Secondly, nation-states are likely to develop biological warfare capabilities when they recognize their

inferiority in conventional capabilities compared to their enemies. Finally, the attractiveness of BW as a form of combat power is enhanced when there is socio-political acceptance of unconventional forms of warfare--or at least a lack of international condemnation over its use.

1945 to 1972

The end of World War II witnessed critical reevaluations of the existing biological warfare programs. Some nations, such as Germany and Japan, saw the dismantling of their programs. Others, such as the US and the Soviet Union, continued with further research and development; some of it possibly enhanced by information gained from the former Axis powers' programs.[19] The superpowers' competition in BW came to a delusory end in 1970 with President Nixon's decision to unilaterally destroy the US's stock of biological warfare agents.[20] But prior to this event a number of technical, operational and socio-political developments occurred that required military planners to rethink BW's potential impact on the battlefield.

The biological sciences underwent revolutionary developments during this period; some that are still creating headlines today. One of the most significant developments of this period was the introduction of the field of aerobiology. Aerobiological techniques provided for employment of biological agents similar to the way chemical agents are employed, and not just by insect vectors and poisoning of water and food.[21] Aerosolization of agents enhances the predictability of attacks and obviates the protection from disease that normal hygiene offers. Other technological developments included: new and better prophylaxes, better therapeutic treatments, efficient agent disseminators, aerosol modelling, techniques for enhancing the survivability of aerosolized agents, development of skin-transferral agents, isolation and synthesis of

new toxins, isolation of psycho-active compounds, genetic engineering, and discovery of novel pathogens (e.g., viroids).[22]

During the late 1950's military planners appear to have become aware of the operational attractiveness of two traits possessed by many biological agents. Non-persistence (neither highly contagious nor likely to remain viable for more than a couple of days in the environment) was recognized to be valuable on a fluid, tactical battlefield. Incapacitating agents (fatalities not expected to exceed 1% to 2%) were recognized as valuable for their potential to consume many of the target forces' resources.[23]

A couple of very important socio-political/global developments occurred during this period. In the 1960's the concept of chemical and biological "... weapons as the poor man's atomic bomb"[24] arose. Throughout the 1950's and 1960's the world's superpowers spent an appreciable amount of resources on research and development in chemical and biological weapons and defenses.[25] The significance of this being that there may still exist considerable stockpiles of agents, materiel, and BW experts that can be exported to other belligerents.

This period also saw a number of accusations of BW proffered against various nations.[26] Between 1947 and 1970 no less than twelve accusations were made of illegal use of biological agents. Not too surprisingly, these accusations reflected the contemporary East--West political polarization of the world. While all of these allegations received considerable press coverage, none of the accusers appeared capable of providing incontrovertible proof to back their charges.

The post World War II era characterizes both the steam-roller effect of modern technological advancements, and the fruitlessness of making allegations without the solid, empirical data necessary to unquestionably prove the use of BW. The techno-

logical advances made between 1945 and 1972 made it possible to safely employ biological agents in a variety of ways, and with a reasonably high degree of predictability, against another force to produce consistently reproducible effects. A spin-off of the technological advancements was the realization that operationally desirable effects can be obtained with ABOs; namely, incapacitation and non-persistency. The many accusations of BW made during this period highlight BW's political sensitivity, and the requirement for immediate, irrefutable evidence from the attack site to prove illegal conduct.

Implications For The Present

Since 1972 advancements in the biological sciences continue at a rapid pace, delivery means for biological agents continue to proliferate around the globe, and regional socio-political patterns that support the employment of ABOs continue to develop. Technological issues and delivery systems will be discussed in detail in Chapter 4, but I would like to briefly discuss current socio-political factors here.

In the past 22 years there have been a number of developments that seem like echoes from the past. These developments include: illegal use of chemical and biological agents, the lack of effective international condemnation and reprisals against the employers of chemical and biological agents, and cause for BW-capable nations to resort to use of unconventional weapons.

In the late 1970s and throughout the 1980s there were both alleged and confirmed chemical/biological attacks. In the Fall of 1979 an unusually virulent anthrax epidemic struck the Russian city of Sverdlovsk.[27] The fatality rate is estimated to have been 30 - 40 fatalities a day for a month, with a total of about 1,000 deaths.[28] But even the combination of this unusual epidemiology with solid human and satellite

intelligence was not enough to gain international consensus over existence of an illegal BW program.

Throughout the late 1970s and early 1980s reports of Soviet use of biochemical warfare in Laos, Kampuchea, and Afghanistan surfaced in the international press.[29] On 13 September 1981, US Secretary of State Alexander Haig declared that physical proof of Soviet employment of mycotoxins in those countries had been obtained.[30] Biochemical casualties in Laos, Kampuchea and Afghanistan were estimated to be as high as 10,000 by 1982.[31] As with the Sverdlovsk incident, international consensus on illegal use of chemical/biological agents could not be obtained, and effective international condemnation has never happened.

The use of Iraqi chemical agents (tabun, sarin, soman and mustard) against Iranians in the Iran-Iraq war resulted in an estimated 50,000 Iranian casualties.[32] Iran's attempts to bring international pressure to bear against Iraq resulted in slow and questionably effective responses by the UN and other world powers. Iraq continued to use chemical agents till the later part of the decade.[33]

The early 1990s saw the crumbling of one of the two global superpowers--the Soviet Union-dominated Communist Bloc. The dissolution of this global power meant the loss of a moderating influence on the nation-states that had been within its sphere of influence. Now only the US remains as a global superpower. Putting debate over our role as "globo-cop" aside, US forces are unarguably the forces most likely to be sent to regional trouble spots to restore peace. Fortunately, we will usually have over-whelming conventional force superiority over our adversaries. Unfortunately, our adversaries are keenly aware of that conventional force imbalance (especially since the Persian Gulf War of 1990 - 1991), and so may feel the same requirement to use weapons of mass destruction that previous belligerents have to ensure their survival.

The pattern of technical, and socio-political developments since the mid-1970s is remarkably similar to those patterns witnessed during the first half of this century. We will be egregiously negligent in our responsibility to protect our forces if we do not acknowledge the threat indicated by these developments, and take the necessary steps to defeat the threat.

Endnotes

[1]Robin Clarke, The Silent Weapons (New York: The David McKay Company, Inc., 1968), 13.

[2]Ibid., 13.

[3]Ibid., 14.

[4]Erhard Geissler, Biological And Toxin Weapons Today (New York: Oxford University Press, 1986), 7.

[5]Clarke, 15.

[6]Geissler, 8.

[7]Ibid.

[8]Ibid.

[9]Ibid.

[10]Clarke, 15.

[11]Ronald M. Atlas, Microbiology: Fundamentals and Applications, 2nd ed. (New York: Macmillan Publishing Company, 1988), 11.

[12]Clarke, 17.

[13]Peter Williams and David Wallace, Unit 731: Japan's Secret Biological Warfare In World War II, (New York: The Free Press, 1989), 9.

[14]Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, Vol. 1, The Rise Of CB Weapons, (New York: Humanities Press, 1971), 116.

[15]Ibid., 5.

[16]US Army, FM 27-10, The Law Of Land Warfare, with change 1, (Washington, D.C.: Headquarters, Department Of The Army, 1956), 18.

[17]Williams and Wallace, 5 - 7.

[18]Ibid., 7.

[19]Ibid., 140, 160 - 163, 191.

[20]Robert Harris, and Jeremy Paxman, A Higher Form Of Killing: The Secret Story Of Chemical And Biological Warfare, (New York: Hill and Wang, 1982), 211.

[21]SIPRI, vol. 1, 123 - 124.

[22]Ibid., 266 - 320.

[23]Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, Vol. 2, CB Weapons Today, (New York: Humanities Press, 1973), 123 - 125.

[24]Ibid., 153.

[25]Ibid., 165 - 168.

[26]SIPRI, vol. 1., 224 - 230.

[27]Elisa D. Harris, "Sverdlovsk And Yellow Rain: Two Cases Of Soviet Noncompliance?" International Security 11 (Spring 1987), 45 - 47.

[28]Ibid., 48.

[29]Ibid., 57.

[30]Ibid., 58.

[31]Joseph D. Douglass, Jr., and Neil C. Livingstone, America The Vulnerable: The Threat Of Chemical/Biological Warfare, (Lexington, Massachusetts: Lexington Books, 1987), 147.

[32]Anthony H. Cordesman, "Creating Weapons Of Mass Destruction," Armed Forces International 126 (February 1989), 54 - 57.

[33]Ibid., 57.

CHAPTER 3

RESEARCH DESIGN

Research Phase I: Compilation Of Available Data And Tools

The data and modelling tools used in support of this study have been obtained from a broad range of open literature. The reason for using a variety of publicly available sources is two fold. First, avoiding the use of classified literature ensures the widest possible dissemination of the data, analyses and findings that are the products of this study. Secondly, there is no single, comprehensive, authoritative work on tactical biological defense. The sources used for this study range from purely academic treatments of putative biological agents and public health concerns, to journalistic reporting of alleged uses of biological agents, to government studies made in direct support of biological defense programs. To the extent possible empirical analyses of the data have been made, but where empirical measures are not available I have used historical events, military doctrine, and logical analyses.

In the process of collecting and initially reviewing the extant literature, I decided that it was necessary to provide a review of the history of BW, and Chapter 2 is the result of that decision. I believe this historical perspective is necessary for several reasons. First, an analysis of the history of BW reveals important technical, battlefield, and socio-political patterns that support a belligerent's use of BW. Second, those same historical patterns are being repeated today. And that is a very important reason for the US military to take proactive steps to deter and/or mitigate offensive use of agents of biological origin (ABOs) by our adversaries. Awareness of the global and regional

conditions that support the use of BW facilitates understanding of the more technical analyses that follow.

As a whole, the current literature provides a significant amount of data which is useful at the tactical level. However, the data are spread throughout a number of publications, and in most cases need to be reanalyzed for use at the tactical level. In other words, a bridge must be made between the current data and the current needs.

The following sections in this chapter address how I will take the available data, apply analytical tools, and synthesize tools that will be immediately useful to the chemical defense officer. These tools will assist the chemical defense officer in intelligence preparation of the battlefield (IPB), planning for operations in a biological threat area, planning for detection and identification operations, and performing BW vulnerability analyses.

Research Phase II: Evaluation Of Threat Agents And Their Tactical Employment

This phase objectively evaluates for tactical employment all the putative biological warfare agents listed in Appendix A, and possible methods for their employment. In addition to evaluation of characterized ABOs (those listed in Appendix A), this research phase also briefly reviews the applicability of less well characterized biologics (e.g., genetically engineered organisms, and novel biochemical substances) to tactical use. Analysis of agents for tactical use yields important information for intelligence preparation of the battlefield, and for vulnerability analysis of friendly forces. Analysis of delivery means also contributes to development of intelligence tools, and accurate vulnerability assessment. Of course, before putative ABOs can be evaluated for tactical employment, some set of criteria are necessary. My first task, then, has been to establish a set of workable criteria for tactically employed agents.

Using The Criteria To Assess The Threat. The number of known disease causing organisms is substantial, but by using some evaluative criteria the number which may reasonably be used as weapons can be significantly reduced. But the existing criteria reflect the conventional views that biological weapons were most likely to be employed at the strategic level. I will base my analyses on a set of criteria given in Erhard Geissler's work Biological And Toxin Weapons Today,[1] (the criteria are listed in Chapter 4).

Other works that I have used in both evaluating these criteria and specific ABOs for their applicability to tactical employment include the following. The Stockholm International Peace Research Institute's (SIPRI's) multi-volume work The Problem Of Chemical And Biological Warfare is an excellent source of objective analyses based on thorough review of the applicable literature up to and including the early 1970s. The 1969 report to the United Nations by the United Nations Group of Consultant Experts on Chemical and Bacteriological (Biological) Weapons is a concise, but remarkably complete, review of chemical and biological warfare's technical aspects although it is focused at the strategic level. Another government publication (from Canada's Defense Research Establishment Suffield) that lends valuable data to this phase is Cher-wonogrodzky and Di Ninno's publication Vaccines, Passive Immune Approaches And Treatment Of Biological Agents. Although its name appears to limit it to medical issues, it actually provides useful operational information. Two historical works which provide significant support to this research are Williams and Wallace's Unit 731: Japan's Secret Biological Warfare In World War II, and Harris and Paxman's work A Higher Form of Killing: The Secret Story Of Chemical And Biological Warfare. Both works provide invaluable historical perspectives on operational concerns in chemical

and biological warfare. Finally, a variety of works authored and co-authored by Joseph D. Douglass Jr. provide important data sources.

A Qualifying Statement. I have refrained from performing an indepth analysis of genetically engineered organisms' impact on belligerents' BW arsenals. I have made this choice for two reasons. First, the offensively oriented products of genetic engineering, if there are any, are not well characterized in the open literature. Second, I do not believe that the state-of-the-art has reached a point where scientists can produce pathogens better than nature can. As a case in point, one only has to consider the sudden, and lethal, outbreak of Hantavirus Pulmonary Syndrome in the Western US during May to June 1993.[2] The pathogen's normal infective route is via the respiratory tract, and it manifests its effects within 2 to 10 days--any genetic engineer would be hard pressed to match the weapon potential of this product of random mutation.

Delivery Systems

The purpose of this section is two-fold: to provide the reader a review of the capabilities of different delivery systems; and to identify systems that may be indicators of a pending biological attack. The types of delivery systems that I include in my analysis are: artillery, ballistic missile and rocket, aircraft, and ground-based generators. The reader will note that all of the delivery systems included for analysis ultimately disperse their payloads through aerosolization. Aerosols have several characteristics which make them desirable for offensive employment of biologics. These include the susceptibility of the respiratory tract, the large area that can be covered in a single attack, and the fact that normal hygiene measures do not prevent respiratory infection/intoxication.[3]

Both historical literature and current articles will make up the data sources for this section. Primary sources of data include: Anthony H. Cordesman's book Weapons

Of Mass Destruction In The Middle East; Fess and William's work Third World Tactical Ballistic Missiles: A Strategy For Defense (U); SIPRI's work The Problem Of Chemical And Biological Warfare, vol. 2, CB Weapons Today; Theodor Rosebury's work Peace Or Pestilence: Biological Warfare And How To Avoid It; and Harvey J. McGeorge's article "Bugs, Gas and Missiles."

These analyses identify the agents most likely to be employed at the tactical level, and the range of biological "payloads" capable of delivery. The next step is to refine the aerosol threat, and analyze the downwind hazard presented by some likely biological attacks.

Research Phase III: Modeling Of Tactical Biological Attacks

Before one can fully understand how best to defend against a biological attack, one must have some understanding of how biological agents will behave in the environment. This phase examines the behavior of ABOs released into the environment using mathematical models developed for downwind dispersal prediction of aerosols.

Delimiting The Form Of Attack

As stated in Chapter 1, I will not consider the use of biological vectors in this study. I will further limit the scope of this study to analyzing the downwind hazard of a point-source generated aerosol. If US forces have air superiority in the area of operations (which is a likely condition), it would be particularly difficult for our adversary to use combat aircraft for ABO delivery. Smaller aircraft, such as unmanned drones and cruise missiles, could theoretically be used in a line-spray fashion. But I have not been able to find anything to indicate that these systems have a spray capability versus just a bursting capability. Finally, time limitations prevent me from extending my research to cover this type of attack.

Standard Values Used For Selected Variables

The mathematical models that are used for downwind hazard prediction are influenced by environmental variables such as temperature and atmospheric pressure. For simplicity and general application I have chosen to use a temperature of 20° Centigrade and an atmospheric pressure equal to 1 atmosphere (atm) or 760 mm Hg (standard sea-level pressure). These values are routinely used in general, demonstrative applications as a matter of scientific convention. Also, these values tend to give worst case results from the calculations; which ensures safe-siding in the models. Other variable values are shown where the models are applied.

Defining The Respiratory Threat

Because the aerosol delivery mode is central to this thesis, it is necessary to have a basic understanding of how the human respiratory system and aerosolized biologics interact. The information presented in this section is primarily from William C. Hinds' text Aerosol Technology: Properties, Behavior, And Measurement Of Airborne Particles,[4] with some data from the United Nations' publication Chemical And Bacteriological (Biological) Weapons And The Effects Of Their Possible Use,[5] and the Army's Final Programmatic Environmental Impact Statement: Biological Defense Research Program.[6]

Optimum Particle Size(s) For Downwind Travel

Settling Velocities Of Aerosolized Particles

The chemical defense officer will use aerosol particle settling velocities to roughly estimate the extent of downwind biological contamination. Establishing the maximum downwind distance based on settling velocities provides a limited range to use in conducting the more complicated follow-on calculations used to refine the downwind

hazard. The following formula is used to calculate the settling velocities (V_{TS}) of aerosolized particles[7]:

$$V_{TS} = \frac{\rho_p d^2 g C_c}{18\eta}$$

Where: ρ_p is the density of the material being aerosolized in g/cm^3
d is the particle diameter in cm
g is the acceleration of gravity; which for all calculations in this thesis is 980 cm/sec^2 (acceleration of gravity at sea-level)
η is the viscosity of air; which at 20° C is 1.81×10^{-4} g/cm•s
C_c is the Cunningham correction factor given by the following formula:

$$C_c = 1 + \frac{\lambda}{d}\left[2.514 + 0.8 \exp\left(-0.55 \frac{d}{\lambda}\right)\right]$$

Where: λ is the mean free path for air; which at 1 atmosphere and 20° C is 0.066 μm.
exp is the exponential function e^x, e = 2.71828

Mathematical Modelling Of Downwind Dispersion

Downwind Dosage Calculation Model

The following series of formulae are used to examine the actual downwind hazards presented by a variety of biological attack scenarios. The value of these analytical tools is that they show the effects of environmental conditions, payload quantity, and agent hardiness on the downwind hazard. Also, these models allow chemical defense officers to perform meaningful vulnerability analyses of their units in a variety of situations.

A simple, but important, formula that for accurate downwind hazard modelling is worth mentioning here. The formula is for determining total respiratory intake (I_t), which indicates how many ABO units are respired at any given agent aerosol concentration.[8] This formula is used to adjust the dosage value (D) in the next formula.

$I_\tau = DR$

Where: I_τ is total respiratory intake in units (pfu, cfu or μg)
D is Dose in units•min•m^{-3}
R is respiratory minute volume in m^3•min^{-1}

The following formula is used to predict dosage (D) given as units•minute•m^{-3} where units are: colony forming units (cfu) for bacteria and fungi, plaque forming units (pfu) for viruses, or μg for toxins. The formula is based on Daniel Wu and Dale Sloop's K-theory model.[9]

The K-Theory Model

$$D = \frac{Q}{2\pi\sigma_y\sigma_z u} \exp\left(-\frac{y^2}{2\sigma_y^2}\right)\left\{\exp\left[-\frac{1}{2}\left(\frac{z-H}{\sigma_z}\right)^2\right] + \exp\left[-\frac{1}{2}\left(\frac{z+H}{\sigma_z}\right)^2\right]\right\}$$

Where: Q is the source strength in the appropriate units (cfu, pfu, or μg)
u is the variable wind speed in x (down-wind) direction in m/min
σ_y is standard deviation of concentration distribution in y direction (see below for calculation)
σ_z is standard deviation of concentration distribution in z direction (see below for calculation)
y is the distance in the cross-wind direction in meters
z is the distance in the vertical direction in meters
H is the height of burst or line release height in meters

Diffusion and Meteorological Parameters for K-Theory Model[10]

Table 1.--Diffusion and Meteorological Parameters for K-Theory

Terrain Stability	Mixing Height (m)	α^1	β^2	SY100	SZ100
Open					
Stable	150	0.64	0.82	2.7	3.25
Neutral	600	0.78	1.10	7.0	5.0
Unstable	3000	0.88	2.08	16.0	14.0
Urban					
Stable	200	0.80	3.7	3.7	5.0
Neutral	500	1.04	13.5	13.5	7.2
Unstable	1000	1.03	21.5	21.5	18.0

[1]Crosswind diffusion coefficient.
[2]Vertical diffusion coefficient.

Calculations For Diffusion Coefficients:

$$\sigma_y = SY100\left(\frac{x}{100}\right)^{\alpha}$$

$$\sigma_z = SZ100\left(\frac{x}{100}\right)^{\beta}$$

Where: x is the distance (in meters) in downwind direction

Correction For Biological Decay

A correction factor must be applied to the value of D to compensate for the loss of the agents' biological activity as they are exposed to the environment. The value obtained for D above will be treated so:

$$D_d = D(\exp(-kt))$$

Where: D_d is the dose given as viable units•minute•m^{-3}
k is the decay constant given as min^{-1}
t is the time time of travel given as downwind distance traveled (m)/wind speed($m•min^{-1}$)[11]

The study up to this point provides the chemical defense officer some very important instruments. These instruments include tools for determining which agents are likely to be employed at the tactical level, intelligence preparation of the battlefield (IPB), and accurate assessment of downwind hazards and friendly forces' vulnerabilities. At this point, enough analyses have been conducted to allow the chemical defense officer to judge ABOs most likely to employed at the tactical level, how and in what quantities the ABOs will be delivered, and the conditions that will support offensive employment of ABOs. The next phase of research is just as critical to a comprehensive biological defense plan--detection and identification of biological attacks.

Research Phase IV: Detection Strategies

This phase of the study investigates how modern detection technologies may assist in location and identification of known markers of biological warfare activities; and how these technologies may be integrated to form a comprehensive, effective battlefield detection and identification plan. The framework for this analysis is based primarily on SIPRI's work The Problem Of Chemical And Biological Warfare, vol. 6, Technical Aspects Of Early Warning And Verification. Other principle data sources include: John Kenkel's publication Analytical Chemistry For Technicians; Silverstein, Bassler and Morrill's Spectrometric Identification Of Organic Compounds, 3d ed.; articles by Charles Murray, Ulf Ivarsson, and Stuart Nichol, *et. al.*; and briefing materials from the May, 1992 Tri-Service Technology Workshop On Biodetection Systems.

Endnotes

[1]Erhard Geissler, "A New Generation Of Biological Weapons," Biological And Toxin Weapons Today (New York: Oxford University Press, 1986), 21-22.

[2]James M. Hughes, C. J. Peters, Mitchell L. Cohen, and Brian W. J. Mahy, "Hantavirus Pulmonary Syndrome: An Emerging Infectious Disease," Science 262 (5 November 1993): 850 - 851.

[3]United Nations Group of Consultant Experts on Chemical and Bacteriological (Biological) Weapons, Chemical And Bacteriological (Biological) Weapons And The Effects Of Their Possible Use, (New York: United Nations, 1969), 19.

[4]William C. Hinds, Aerosol Technology: Properties, Behavior, And Measurement Of Airborne Particles (New York: John Wiley & Sons, 1982), 211 - 231.

[5]United Nations Group of Consultant Experts, 60 - 61.

[6]US Army Medical Research And Development Command (USAMRDC), Final Programmatic Environmental Impact Statement: Biological Defense Research Program, (Fort Detrick, Frederick, Maryland: US Army Medical Research And Development Command, 1989), A9-22.

[7]Hinds, 40 -45.

[8]Ibid.

[9]Daniel Wu, and Dale W. Sloop, A Package of Transport And Diffusion Models For Biological And Toxin Agents, CRDEC Technical Report No. 86034 (Aberdeen Proving Ground, Maryland: U.S. Army Chemical Research, Development and Engineering Center, 1986), 12 - 13.

[10]Wu and Sloop, 70.

[11]US Army Medical Research And Development Command (USAMRDC), A9-21 - A9-22.

CHAPTER 4

ANALYSIS

Analysis Of Potential Agents Using Established Criteria

This section analyzes the applicability of various ABOs to tactical employment using existing criteria; criteria which were designed to evaluate the effectiveness of ABOs in all applications (strategic, tactical and terrorist). Because these criteria have such general application, I will also discuss each criterion's relevance to tactical operations, as well as how each criterion may be used in planning for force protection. For simplicity's sake, I will use "attacker" to designate the belligerent making offensive use of biologics, and "defender" to designate against whom the biologics are used. I will use Erhard Geissler's list of criteria[1] as an outline for discussion.

Analysis Of Potential Agents

1. The agent should consistently produce a given effect: death or disease

First, I would like to clarify the definition of disease for purposes of this analysis. Disease, as is commonly known, connotes sickness and impairment of ability to perform work. In Chapter 1, I defined incapacitating agents as ABOs which are capable of rendering persons incapable of performing their normal duties and which are not likely to produce fatalities. For continuity I will use the term incapacitating agent in place of disease.

Consistency of effects is a desirable trait in any weapon, and so it's only logical to [illegible]or consistency in the types of casualty effects that biological weapons will

yield. Furthermore, in tactical operations, the casualty effect that is most desirable is incapacitation.

An ABO which is an incapacitating agent has at least two advantages over lethal agents in tactical employment.[2] First, a large number of ill soldiers will place a greater burden on the defender's logistic system than a large number of deceased soldiers. Transportation assets, hospital facilities, and antibiotics/antitoxins will be consumed at a tremendous rate. Second, if the target area is in close proximity to the attacker's own positions, and the attacker is concerned with back-drift of ABOs, then the attacker may prefer to employ an incapacitating agent over a lethal agent. Of course, the attacker will also have to treat his affected solders, but the number of casualties should be relatively low (especially if the attacking force has been immunized), and the burden on the attacker's logistic system acceptable. An incapacitating agent would be especially desirable when the target area contains non-combatants.

Historical examples of incapacitating agents' appeal can be seen in the American military's interest in brucellosis, and in the former Soviet Union's doctrine. The characteristics of brucellosis which make it useful include: debilitating effects, low mortality rate, and high infectiousness. Brucellosis' symptoms include "chills and . . . fever, headache, loss of appetite, mental depression, extreme exhaustion, aching joints and sweating."[3] In extreme cases its effects may be felt for up to a year.[4] Even though brucellosis has a low mortality rate (2 - 6%), it is very infectious (only about 10 viable organisms are required to initiate an infection).[5] The value of incapacitating agents is underscored by the former Soviet Union's doctrine, which had written in use of incapacitants in exercise scripts.[6]

There are two special cases on the tactical battlefield where I can see that an attacker may choose to use lethal agents over incapacitating agents. Instead of just temporarily defeating a critical command and control node, an attacker may choose to

impact it to a greater degree by using lethal agents. Critical lodgement areas with high troop concentrations may be another lucrative target for lethal agents.

Table 2.-- Incapacitating Agents Of Biological Origin

Causative Agent	Effect	Mortality Rate (%)
Brucella spp.	Brucellosis	2 - 6
Coccidioides immitis	Coccidioidomycosis	0 - 50
Coxiella burnetii	Q-fever	1 - 4
Rickettsia typhi	Murine or Endemic typhus fever	0 - 2
CHIK	Chikungunya fever	0 - 1
Dengue Virus	Dengue or Breakbone fever	1 - 20
Influenza Virus	The flu	0 - 1
RVF	Rift valley fever	1 - 10
VEE	Venezuelan Equine Encephalitis	0 - 2
Aflatoxin B	Fungal-related food poisoning, probably similar to T-2 poisoning	Unknown
Staphylococcus Enterotoxin B (SEB)	Acute food poisoning symptoms	Unknown
T-2 toxin	Skin irritation, nausea, diarrhea	Unknown

Sources: Appendix A, and information in SIPRI's work The Problem Of Chemical Biological Warfare, vol. 2 CB Weapons Today, (New York: Humanities Press, 1973), 122.

2. The concentration of the agent needed to cause death or disease -- the infective dose -- should be low

The reasoning behind this criterion is pretty evident. The smaller the quantity of agent needed to produce the desired effects, the fewer delivery resources (e.g., aircraft, and artillery pieces) required, and the less the cost to the attacker. But this argument is only valid for biotoxins (in which case "infective dose" would be replaced with "intoxicating dose"); not for microbial ABOs.

The number of effective doses (i.e., one effective dose will produce the desired effect of debilitating infection or intoxication in a person with at least 50% probability)

that can be packed into one gram of microbial material is substantial. Consider the case of anthrax, which has a relatively high infective dose. The number of organisms that may be contained in 1 gram of wet agent has been estimated to be approximately $3x10^{10}$.[7] The effective dose is about $1.3x10^{3}$. A single gram of wet anthrax preparation could theoretically infect, with 50% probability, 23 million persons. Of course, that number assumes that each person would inhale exactly 1300 anthrax organisms, and that none of the organisms would be lost in the environment. This example demonstrates that for microbial ABOs the efficiency of dissemination becomes a more critical factor than the infectious dose.

For toxins, however, the effective dose may be a concern to a belligerent who has the option of using either chemical agents or biotoxins. I will examine the relative efficacies of lethal and incapacitating biotoxins to chemical agents in the next two paragraphs.

I have chosen to use nerve agent sarin (GB) as the chemical "yardstick" to measure lethal biotoxins against because of its relatively low lethal dose, and because its primary route of intoxication is through the respiratory route (as with most biotoxins). Using lethal dose data for GB for a person at rest (100 $mg \cdot min/m^3$),[8] an inspiration rate for a person at rest (0.0084 m^3/min),[9] and a body mass of 158.6 pounds (72 kg), one can compare the casualty producing effectiveness of lethal chemical agents versus lethal biotoxins. As it turns out, lethal biotoxins which have an LD_{50} greater than 12 $\mu g/kg$ are no more "efficient" at producing casualties than are standard nerve agents.

For incapacitating agents, I have chosen the chemical agent CNS for comparison. CNS produces choking, tearing, vomiting and lung damage (the effects can last for weeks), and has an effective dose of only 60 $mg \cdot min/m^3$.[10] The same

inspiration rate and body mass were used as for lethal agents. For incapacitating agents, an "efficient" biotoxin would have an effective dose of 7μg/kg or less.

At this point, the belligerent who has the option of using either chemical agents or ABOs would have to consider other factors, such as: ease of manufacture, ease of protecting the attacking force, and vulnerability of the defending force (e.g., inability to detect the agent, inadequate protection or inadequate medical treatment). Table 3 lists those biotoxins which are more toxic than corresponding chemical agents.

Table 3.--Biological Toxins With Low Effective Doses

Toxin (source & type)	Lethal or Incapacitating Dose (μg/kg)
Botulinum (bacterial neurotoxin)	0.00003 - 0.01
Diphtheria toxin (bacterial cytotoxin)	0.03
Staphylococcus Enterotoxin B (bacterial incapacitating agent)	0.04
Palytoxin (coral neurotoxin)	0.08 - 0.4
Batrachotoxin (frog neurotoxin)	0.1 - 2.0
Nivalenol (fungal cytotoxin)	0.4
Taipoxin (snake neurotoxin)	2.0
Beta-Bungarotoxin (snake neurotoxin)	2.0
Ricin (plant cytotoxin)	3.0
Conotoxin (snail neurotoxin)	3.0 - 6.0
Alpha-Latrotoxin (spider neurotoxin)	10.0
Tetrodotoxin (puffer fish neurotoxin)	10.0
Saxitoxin (algal neurotoxin)	10.0

3. The agent should be highly contagious

At the tactical level, contagiousness is not a particularly desirable trait. A highly contagious agent may lead to an epidemic. While a planned epidemic may be desirable at the strategic level, where the target population is located on a separate continent,[11] an attacker is not likely to want to produce an epidemic within the same region where his own forces and/or civilian population are. Even if an attacker has been able to vaccinate a large proportion of his forces and civilian population against

the communicable ABO, a risk remains. Not only will individuals differ in their immunological responses to vaccines, but the overall efficacy of vaccines may be questionable,[12] and their effectiveness may last for only limited durations.[13]

The agent will in effect become "persistent."[14] The initial round of infection, incubation, disease manifestation and communication will be followed by repeated rounds until appropriate medical treatment can be administered to the affected population(s). Hence, the disease will persist in the target population until appropriate treatment has been accomplished. Also, some communicable diseases have natural non-human reservoirs, which may greatly complicate control of the disease.

The first two reasons show the difficulty in controlling communicable diseases. This leads to the final reason why highly communicable diseases are not well suited for tactical employment. Communicable diseases and the epidemics they are likely to create are very unpredictable. Influenza (flu) illustrates the problems associated with the control of epidemics. Flu epidemics occur nearly annually, and even with the best epidemiological tools available in a peace-time environment several hundred flu-related deaths occur in the US each year.[15] The influenza virus, like other highly communicable diseases, can also infect other animals.[16] This complicates its control, and increases its chances of mutating into immunologically new strains. As a panel of chemical and biological warfare experts have noted, "The history of epidemiology is rich with surprises."[17]

While contagiousness may be a favorable trait in a strategically employed ABO, where the attacker wants to inflict maximum damage with minimum assets and against a distant target, it is not desirable for tactical employment. Table 4 lists those microbial ABOs which are highly contagious, and so could present significant problems for the tactical employer of BW.

Table 4.--Highly Contagious ABOs

Causative Agent	Disease	Remarks
Chlamydia psittaci	Parrot fever or ornithosis	Birds are also reservoirs of this disease
Legionella pneumophila	Legionnaires Disease	
Rickettsia provazekii	Infectious or classic typhus fever	Contagiousness depends on low level of sanitation; transmitted via the body louse
Shigella spp.	Dysentery	Contagiousness depends on low level of sanitation
Vibrio cholera	Cholera	Contagiousness depends on low level of sanitation
Yersinia pestis	Plague	Other reservoirs (hosts) include rodents (including domestic animals) & fleas
Influenza virus	Flu	Type A influenza also infects many species of mammals and birds
Variola virus	Smallpox	Immunization programs have been dramatically reduced since the late 1970's

Sources: Ronald M. Atlas, Microbiology: Fundamentals and Applications, 2nd ed. (New York: Macmillan Publishing Company, 1988), 622, 666, 699.
Robin Clarke, The Silent Weapons, (New York: The David McKay Company, Inc., 1968), 80, 250 - 251.
Heinz Fraenkel-Conrat, Paul C. Kimball, and Jay A. Levy, Virology, 2nd ed. (Englewood Cliffs, New Jersey: Prentice Hall, 1988), 141.
Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, Vol 2, CB Weapons Today, (New York: Humanities Press, 1973), 122.

4. The agent should have a short and predictable incubation time from exposure to onset of the disease symptoms

Before analyzing this criterion I will first review current, applicable doctrine. FM 100-5, Operations, stresses that we are likely to be involved in regional conflicts, and so must be prepared to deploy and fight across the globe.[18] This global projection force mission means that we must be ready to deploy to austere theaters, and rapidly build up our combat strength to allow us to decisively and quickly overcome the enemy. FM 100-17, Mobilization, Deployment, Redeployment, Demobilization, provides the following deployment objectives:

> The lead brigade of [the deploying contingency] force projected for combat operations will be capable of being on the ground by C+4 (airlift) [C-day is the day strategic movement begins], the lead division by C+12 (airlift), and two heavy divisions deployed . . . by C+30 (air/sealift). By C+75 the full corps (remaining two divisions), with its support command (COSCOM) and appropriate echelons above corps (EAC) logistics above the corps will be on the ground.[19]

It is apparent that the majority of troops will arrive in theater between C+12 and C+75, and their main missions will be defense of the lodgement area and preparation for combat operations. This window of more than 60 days would be a logical opportunity for an attacker to employ ABOs. Appendix A shows that none of the potential ABOs have a latent period greater than 60 days. If an attacker chooses to employ BW during the deployment phase, this criterion will not weigh in the decision of which biologic(s) to use.

However, it is equally likely that an attacker may choose not to employ ABOs early in the conflict (i.e., during the deployment phase) for fear of harsh reprisal. But if conditions evolve to the point of impending defeat, then the attacker may choose to employ biologics in the hopes of gaining a combat power advantage over the defender. In this case, rapid manifestation of disease would be necessary. The most rapid acting ABOs are the toxins, all of which manifest their effects within 12 hours; most in less than 4 hours. Toxins would be the logical choice in the fast-paced scenario that was envisaged in a conflict with the former Soviet Union. But in a slower paced operation (either due to restrictive terrain, or because of closely matched combat ratios between belligerents), microbial agents could be effectively employed.

Table 5 is a list of microbial agents that relatively rapidly manifest their effects. I have chosen a latent period of 7 days or less to characterize "relatively rapid." I believe this to be a reasonable duration to conduct delaying operations and allow the pathogens to manifest their effects.

Table 5.--Microbial Agents With Relatively Rapid Effects

Causative Agent	Disease	Latent Period (Days)	Remarks
Bacillus anthracis	Anthrax	1 - 4	easy to produce
Francisella tularensis	Tularemia	1 - 10 (ave = 3)	can be produced in quantity, but with difficulty
Yersinia pestis	Plague	1 - 4	easy to produce
Vibrio cholerae	Cholera	1 - 5	allegedly produced in quantity by Japan in W. W. II
Pseudomonas mallei	Glanders	2 - 14	easy to produce
Pseudomonas pseudomallei	Melioidosis	1 - 5	easy to produce
Shigella spp.	Dysentery	1 - 3	easy to produce
Rickettsia rickettsii	Rocky Mountain Spotted Fever	2 - 14 (ave = 7)	requires tissue culture
Chlamydia psittaci	Parrot Fever or Ornithosis	1 - 4	requires tissue culture
CHIK	Chikungunya fever	2 - 6	requires tissue culture
VEE	Venezuelan Equine Encephalitis	1 - 6	requires tissue culture
Dengue Virus	Dengue or Break-bone Fever	2 - 7	requires tissue culture
YFV	Yellow Fever	1 - 6	requires tissue culture
CCHFV	Crimean-Congo Hemorrhagic Fever	3 - 6	requires tissue culture
RVF	Rift Valley Fever	1 - 5	requires tissue culture

Sources: C. H. Collins, Patricia M. Lyne, and J. M. Grange, Collins and Lyne's Microbiological Methods, 6th ed. (Oxford: Butterworth & Heinemann, 1989), 241, 265, 292.
Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, Vol. 2, CB Weapons Today, (New York: Humanities Press, 1973), 64 - 70.
Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, Vol. 1, The Rise of CB Weapons, (New York: Humanities Press, 1971), 114 - 115.

5. The target population should have little or no natural or acquired immunity or resistance to the agent

The value of this criterion is self evident. Using an agent such as smallpox against a force which had recently been immunized against it would be at best useless,

and at worst an invitation to strong reprisals (current smallpox vaccines provide high levels of protection).

6. Prophylaxis against the agent should not be available to the target population

Before discussing the importance of this criterion, I wouldd like to ensure that the reader understands that there are two classes of prophylactic, or preventive, measures. These classes are immunoprophylaxes (vaccines), and chemoprophylaxes (antibiotics, e.g. ciprofloxacin used in Operation Desert Storm for protection against anthrax).

The importance of effective immunoprophylaxes is seen in several statements. Erhard Geissler stated "...the most important impact of genetic engineering for BW is that 'an increased protection capability may be an inducement to use biological warfare, since the instigator has a decreased risk of being harmed by his own actions.'"[20] Cherwonogrodzky and Di Ninno go so far as to proclaim that "...in a theater of war, the victor may be the one, not with the most weapons, but the one with the only vaccine."[21] Another indicator of the importance of vaccines is seen in the Chinese government's decision to classify its vaccine for a strain of brucellosis as "Top Secret."[22] Philip K. Russell, M.D. and former commander of U.S. Army Medical Research and Development Command (USAMRDC) is quoted as saying that countries with biological warfare programs have "...given up on a number of agents because our vaccines are so good."[23] Russell was also cited as stating that our troop immunizations discouraged the Iraqis from employing ABOs.[24] Obviously, knowing whether an attacker has effective vaccines in appropriate quantities for potential ABOs will be an important intelligence requirement (IR).

In addition to prophylaxes' availability, their efficacy and side effects must also be considered. As was mentioned in the analysis of criterion number 3, many vaccines for potential ABOs are of questionable value. So even if a majority of the target pop-

ulation receives the vaccine, only a limited number of vaccinees may gain an effective immunity. Also, there are few prophylaxes which are without undesirable side effects. Some vaccines will induce mild disease symptoms, and some vaccines may actually cause severe reactions (anaphylaxis) in a small number of vaccinees. Chemoprophylaxes can also have undesirable side effects, as well as being expensive to administer on a large scale. Because of prophylaxes' undesirable and sometimes questionable effects, they are typically administered only when a threat is likely; which means that an attacker can inflict considerable casualties on a defender by ensuring attack before protective measures are administered. The start of prophylactic treatments for both defender and attacker will be an item of intelligence value.

The tables in Appendix A show which pathogens vaccines have been developed for. There are only vaccines for 23 of 62 potential ABOs. There are an additional 11 vaccines that are currently under development. The reader will note, however, that there are a considerable number of ABOs for which I have not made an entry in the Vaccine/Toxoid column. This is simply because I do not have current, unclassified data indicating whether or not vaccines exist for these pathogens.

7. <u>The agent should be difficult to identify in the target population, and little or no treatment for the disease caused by the agent should be available</u>

At the tactical level, this criterion should read "The biological attack should be difficult to identify--regardless of whether treatment for the disease is available or not." If criteria 1 and 5 (consistency of effect and no immunity in the target population) are satisfied, then identification of a biological attack is the most important defensive step.

A defender without active biological detection assets will only have unnatural outbreaks of disease to indicate biological attacks. The attack will be complete by the time medical channels identify the pathogen through classical procedures (symp-

tomatology, histopathology, isolation and identification of the pathogen). Further complicating timely defense against biological attacks is the unusual route of infection or intoxication--the respiratory route. Infection or intoxication by aerosolized pathogens causes disease symptoms unlike those found in naturally acquired disease, and so confounds rapid diagnosis by medical personnel.[25]

Once medical agencies for the defender have identified the agent, treatment will begin with intent to reduce the incidence of fatalities, and to mitigate symptoms and shorten convalescence. But without the capability to detect a biological attack in progress, the defending force will be afflicted with productive infections and intoxications before any protective measures can be taken. Also, recall from analysis of criterion 1 that incapacitating agents would logically be the agents of choice at the tactical level, and fatality rates between treated and non-treated targets of incapacitating agent attacks will only differ by a few percent. So even if the defender does possess adequate treatment capabilities, he will be forced to deal with an additional logistical burden that will further degrade his overall combat power.

Analysis of this criterion provides two important considerations for the tactical chemical defense officer. First, effective real-time identification of in-progress biological attacks is critical to timely implementation of protective measures. And second, effective treatments may only shorten the duration of effects and possibly save a few fatalities, but may not significantly protect the defender's combat strength.

8. The aggressor should have means to protect his own forces and population against the agent clandestinely

In the analysis of criterion 6, I provided several citations which testified to the importance of vaccines. In the analysis of this criterion I will discuss prophylaxes' importance to the attacker, the value of implementing protective measures clandestinely, and possible means of clandestine implementation of protective measures.

Vaccines allow attacking forces to operate unencumbered in the vicinity of the target area during attack, and in the biologically contaminated target area shortly after the attack (when residual concentrations of bioactive ABOs are low enough to not overcome the attackers' immunity). The defending force, on the other hand, must either suffer potentially high casualty rates, or don degrading protective clothing. It is worthwhile to note that a U.S. medical journal stated that possible reasons the Iraqis did not employ biological weapons during Operation Desert Storm was not because they lacked the agents and the delivery means, but because they had neither vaccinated their troops nor prepared their medical system to accept BW casualties.[26] Obviously, knowing what diseases the attacking force has been protected against will be vital to the defender in determining which ABOs may be employed by the attacker.

I suggest two possible means that the attacker may use to protect this vital information from the defender. The first means is to use an aerosol vaccine that could be clandestinely delivered to a large population in a short amount of time. Reportedly, the U.S. Army has investigated the feasibility of aerosol immunization, but the results of these studies are unknown.[27] The second method of clandestine protection of forces is conventional administration of prophylaxes coupled with a covering propaganda plan. A well constructed propaganda campaign, which claimed indications of an impending epidemic, could cover sudden, widespread administration of protective measures. The first method promises superior operational security for the attacker, since the vaccinees themselves would be unaware of their vaccinations. But this method requires a sophisticated aerobiology program, and a significant research and development effort. The second method is technologically much more feasible, but is less secure.

Analysis of this criterion suggests several information requirements that may be exploited at the tactical level:

(1) Aerosol vaccinations may be indicated by detection of unusually high concentrations of aerosolized biologics along the fringes of the attacker's positions (samples should be immediately evacuated through medical intelligence channels for laboratory assay of immunogenicity, and bioactivity).

(2) Prisoners of war should have blood samples drawn to determine what potential ABOs the attacking force may have been immunized against (the U.S. allegedly did this with Japanese prisoners during World War II[28]).

(3) Captured enemy items which would indicate preparations for biological warfare, to include: antibiotic tablets, muscle relaxant drugs (e.g., diazepam or valium), and personal immunization records.

(4) Sudden claims by the attacker of impending and new epidemics should be regarded as suspect until confirmed by a neutral agency (e.g., the World Health Organization (WHO)).

9. <u>The agent should be amenable to economical mass production</u>

Unfortunately, definitive, empirical data are not available to compare the costs and times involved in the production of the various potential ABOs. The open literature that does address agent production uses data from the 1940s to 1960s. This limited, dated information has minimal value in attempting to assess current production characteristics. The revolutionary developments of the past couple of decades offer a variety of high efficiency culture methods to belligerents interested in developing a BW program. Technologies that have had, and which promise to have, the most impact on ABO production include: continuous culture techniques, high efficiency tissue culture techniques, biochemical synthesis, and genetic engineering.

The easiest type of agents to produce are the bacteria and fungi. These agents, especially the bacteria, represent the "classical" biological warfare agents that were tested, produced and allegedly employed during the two world wars. These microbes

can be easily grown in large scale using simple, relatively inexpensive equipment. Growth media are typically simple broths of yeast or meat digests, and may or may not contain nutrient supplements (e.g., minerals, amino acids, common proteins).[29] Large volumes of this group of agents may be grown in several days or less. Perhaps the most important development in production of this class of ABOs is the continuous culture technique. Simply stated, this technique is the process of continually adding nutrients and medium while harvesting portions of the product. Advantages include economy and consistency of product. This technique was actually developed in the United Kingdom as a part of their biological defense program during World War II.[30]

The rickettsia, rickettsia-like organisms (i.e., *Chlamydia* and *Coxiella*) and viruses are more difficult to produce in quantity than bacteria and fungi. These intracellular agents require tissue cultures for production, and tissue cultures compare to bacterial and fungal cultures by being slower growing, more expensive and complex, and more reliant on sterile handling procedures. Tissue culture may be relatively basic and just slightly more difficult than bacterial culture (e.g., using whole eggs to grow microbes like *Coxiella burnetii*) or it may be significantly more complex, relying on fastidious mammalian cells. But all types of tissue culture benefit from the recent developments in cell biology and culture technology. Developments in culture media, substrates (most animal cells have to be "anchored" to function properly) and culture vessels allow for propagation of heretofore non-culturable cells, denser cell cultures, and reliable aseptic biological containment.

Biological toxins are typically less economical to produce than either of the two previous classes of biological agents. Not only does the organism which is the source of the toxin have to be grown, but then the toxin has to be extracted from the organism. One only has to consider the size of an average rattlesnake compared to the volume of venom that it produces to gain an appreciation for how difficult toxin production can

be. Some toxins, however, may be produced at relatively reasonable costs, because the toxin is: (1) derived from a microbe that can be grown quickly and in quantity, (2) derived from a plant that produces fairly high concentrations of toxin, or (3) may be synthesized free of the organism it was originally found in. This class of biological agents probably stands to gain the most from genetic engineering techniques. A theoretical application would be to splice the gene for a potent toxin (e.g., the snake neurotoxin taipoxin) into a microbe which grows rapidly and at little expense.[31] The engineered microbe would enable the attacker to produce large quantities of toxins in short periods of time.

Table 6 provides a list of biological toxins that may be considered the most amenable to large scale production. Feasibility is based on the criteria listed above; i.e., microbial or plant origin, or capable of synthesis. I want to stress at this point that large scale production of biotoxins has yet to be recorded in the open literature. Large scale production and purification of biotoxins, regardless of their origin, is still a difficult task.[32]

Table 6.--Biological Toxins With Potential For Large Scale Production

Toxin	Natural Source	Remarks
Abrin	*Abrus precatorius* (jequirity plant)	good recovery from seeds, used in cancer research
Aflatoxin	*Aspergillus flavus* (fungal toxin)	toxin yield highly dependent on growth conditions
Anatoxin A	*Anabaena flos-aquae* (bacterium)	
Batrachotoxin	*Phyllobates aurotaenia* (poison arrow frog)	can be synthesized
Botulinum	*Clostridium botulinum* (bacterium)	standardized BW agent
Cobrotoxin	*Naja naja atra* (cobra snake)	can be synthesized
Diphtheria toxin	*Corynebacterium diphtheria* (bacterium)	used in cell biology studies
Microcystin	*Microcystis aeruginosa* or *M. cyanea*	

Table 6.--Continued

Toxin	Natural Source	Remarks
Nivalenol	*Fusarium nivale* (fungal toxin)	"Yellow Rain" component
Palytoxin	*Palythoa toxica* (coral)	can be synthesized
Ricin	*Ricinus communis* (castor bean)	good recovery from seeds, used in cell studies and cancer treatment
Staphylococcus enterotoxin B	*Staphylococcus aureus* (bacterium)	standardized BW agent
Saxitoxin	*Gonyaulax catanella* or *G. tamarensis* (dinoflagellates)	can be synthesized, used in neurochemical studies
Staphylococcus enterotoxin B	*Staphylococcus aureus* (bacterium)	Staphylococcal poisoning was allegedly used covertly in WW II
T-2	*Fusarium tricinctum* (fungal toxin)	can be synthesized, powerful carcinogen, "Yellow Rain" component
Tetrodotoxin	*Tetraodontidae* (puffer fish)	can be synthesized

Sources: Susan Budavari, Maryadele J. O'Neil, Ann Smith, Patricia E. Heckelman, The Merck Index: An Encyclopedia of Chemicals, Drugs and Biologicals, 11th ed. (Rahway, N. J.: Merck & Co., Inc., 1989), 2, 30, 157, 383, 1052, 1107, 1307, 1330, 1456, 1541. Peter Williams and David Wallace, Unit 731: Japan's Secret Biological Warfare In World War II, (New York: The Free Press, A Division Of Macmillan, Inc., 1989), 123.

10. The agent should be reasonably robust and stable under production and storage conditions, in munitions and during transportation--storage methods should be available that prevent the gross decline of the agent's activity

Unfortunately, I have not been able to find storage life data in the open literature that can be applied to all of the potential ABOs. What I will provide instead is a discussion of what data is available on biological preservation, and a discussion of this criterion's relative importance to a defending force

Biological activity in microbes and proteinaceous toxins relies on the preservation of their constituent protein or peptide chains' three dimensional structure(s). Without getting into a long and tangential discussion on biochemistry, suffice it to say that long term preservation of protein-based biologics is best met through cold temper-

atures (to slow down molecular activity or motion), and minimum water in the immediate environment (to prevent expanding ice crystals from breaking apart the protein chains). Lyophilization, or freeze-drying, is a preservation technique which has come into universal use since World War II for the long-term preservation of biologics. Lyophilized materials are advantageous in biological warfare for another reason. Freeze-dried agents retain their potential for activity at ambient temperatures, which allows more time for removal from storage, to dispersion, to contact with hosts before the agents lose a significant amount of biological activity. A method for moderately long periods of storage is freezing at ultra-low temperatures (usually around the temperature of liquid nitrogen (-196° C)).[33] *Coxiella burnetii* the causative agent for Q-fever, can retain its viability for up to 5 years when stored at just -50° C.[34]

Non-proteinaceous toxins are also susceptible to degradation during long-term storage, but I believe that because of their smaller and more rigid structure they are less susceptible to moderate temperature influences. Unfortunately, I have been unable to find any empirical data to either support or refute my hypothesis.

An important consideration in evaluating the relative importance of this criterion is recognition that the attacker has options available to him. In the analysis of criterion 9, I pointed out that bacterial and fungal agents may be produced in quantity in about a week. If the attacker has enough production materials to produce sufficient quantities for several tactical strikes within a couple of weeks, he may not have to concern himself with storage issues. Conversely, toxins and viruses are difficult and time consuming to produce. If the attacker wishes to employ biological toxins , he may be very concerned with agent shelf-life. Unfortunately, the signatures for biological weapons production and storage facilities are quite small and indistinguishable; especially if the attacker has several weeks time to make up for limited production capabilities.

Analysis of this criterion provides several conclusions: (1) long-term storage of biologics is not a problem, (2) lyophilization is preferable when agents must be moved from storage to munitions filling/dissemination points, and (3) the attacker can avoid any costs associated with storage by using freshly produced microbial agents. In fact, aerosol infectivity tests conducted with *Francisella tularensis* (the causative agent of tularemia) indicate that fresh preparations are more infective than stored cultures.[35]

11. The agent should be capable of efficient dissemination--if it cannot be delivered via an aerosol, living vectors (e.g., fleas, mosquitoes or ticks) should be available for dispersal or [there should be] some form of infected substrate

I mentioned in Chapter 3 that this theisis only considers the use of aerosol employment of ABOs. I have made this decision because biological vectors (e.g., rats, mosquitos, and fleas) have several significant tactical disadvantages.

First, the time required to transmit the disease(s) to the target population would be significantly increased. Consider the events that must occur when fleas are used as vectors for plague: (1) delivery of the insect vectors, (2) time for the fleas to attach to a host (if not the target hosts, then intermediate hosts, which would cause greater delays to affect casualties), (3) infection of the target hosts by the vector, and (4) pathogen incubation within the target host. An example of how long it takes to affect casualties may be seen in an alleged plague attack by the Japanese on the Chinese town of Changteh in November 1941.[36] In this attack, the first casualty did not occur until a week after the airplane-delivered attack, and it was an additional 2 days before any additional casualties were effected. In comparison to this example, with an average latent period of 9 days, aerosol employment is significantly more rapid, having a latent period of only 1 - 4 days.

A second disadvantage to using biological vectors is the increased persistency of ABOs in the target area. Not only will the vectors protect the agents from damaging environmental effects, but the vectors could establish reservoirs for the pathogen. If

reservoirs become established, then only eradication of the infected vectors could remove the pathogen from the area.

Finally, there is the distinct chance that the vectors will drift into the attacker's own forces. Biological vectors, which have the ability to migrate in any direction, virtually eliminate the attacker's ability to predict with any degree of confidence the spread of the ABO(s) employed.

As with other types of offensive data on biologics, the information that I have been able to find on aerosolization of ABOs is dated to the early 1970s. However, there have been a number of biologics added to the list of potential ABOs since then. The class of potential ABOs which are most lacking in aerosolization data are the toxins. The toxins that are considered "standardized" biological weapon agents (i.e., botulinum toxin, staphylococcus enterotoxin B, ricin, T-2 toxin, nivalenol, and saxitoxin) are certainly capable of aerosol employment. Table 7 provides a list of microbial ABOs which are known to be capable of aerosol employment, and indicates those that were considered "standardized" biological weapon agents in 1973.

Table 7.--Microbial Agents Known Suitable For Aerosol Employment

Agent	Disease	Remarks
Bacillus anthracis	Anthrax	Standardized BW agent
Brucella spp.	Brucellosis	*B. suis* is a standardized BW agent
Chlamydia psittaci	Parrot fever	
Coccidioides immitis	Coccidioidomycosis	
Coxiella burnetii	Q-fever	Standardized BW agent
Francisella tularensis	Tularemia	Standardized BW agent
Legionella pneumophila	Legionnaires disease	Natural epidemics occurred in 1968 (Pontiac, MI) & 1976 (Philadelphia, PA)
Pseudomonas mallei	Glanders	Allegedly used by Germans in W. W. I
Pseudomonas pseudomallei	Melioidosis	
Rickettsia prowazekii	Classic typhus fever	
Rickettsia rickettsii	Rocky mountain spotted fever	

Table 7--Continued

Agent	Disease	Remarks
Salmonella typhi	Typhoid fever	Also effective in water and food contamination
Yersinia pestis	Plague	Allegedly employed by the Japanese during W. W. II
CHIK	Chikungunya fever	
Dengue Virus	Dengue or breakbone fever	
Influenza Virus	Flu	
RSSEV	Russian spring-summer encephalitis	
RVF	Rift valley fever	
Variola Virus	Smallpox	
VEE	Venezuelan equine encephalitis	Standardized BW agent
YFV	Yellow fever	Standardized BW agent

Sources: Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, Vol. 2, CB Weapons Today, (New York: Humanities Press, 1973), 38 - 39.
Ronald M. Atlas, Microbiology: Fundamentals And Applications, 2nd ed. (New York: Macmillan Publishing Company, 1988), 658 - 660, 665 - 666.

12. The agent should be stable during dissemination--if it is to be delivered via an aerosol, it must survive and remain stable in air until it reaches the target population.

There are a number of factors that influence how long an ABO will retain its viability or biological activity in an aerosolized state. Some of these factors are directly dependent on the environment, and include: intensity of sunlight, temperature, and ambient humidity. Other factors are determined by the employer of ABOs, and these include: stabilizers in the agent preparation, method of aerosolization (e.g., explosive force, pressure feed, or aspiration), use of wet or dry preparations, and micro-encapsulation of the agents. Potential stabilizers for decreasing aerobiological decay rates include "...certain sugars, polyhydric alcohols [alcohols with more than one -OH group; e.g., ethylene glycol, glycerol, and inositol] and glycerol-thiourea mixtures."[37] The cyclical, polyhydric alcohol inositol has been shown to be particularly effective with some ABOs, and spent growth medium has also been shown to afford some

protection.[38] Dry (i.e., lyophilized) preparations of some agents have been shown to retain their viability longer during aerosolization than wet preparations.[39] Data on potential methods and materials for use in microencapsulation are particularly rare, but I would submit that a likely candidate may be the protein-polysaccharide complex mucin. The Japanese investigated the use of mucin in biological warfare during World War II, and there is a record of an event where a Japanese plane disseminated plague-carrying granules that could have been made of mucin.[40]

Although the available data on aerosol stability represents only a fraction of all the potential ABOs, it does provide some useful data. The reader can see from Table 8 that sunlight rapidly degrades both microbial pathogens and protein toxins. Table 8 also shows that different ABOs' decay rates vary significantly with relative humidity. The tactical chemical defense officer can compare current meteorological conditions with known aerobiological decay data to anticipate which ABOs may be employed by an attacker.

Table 8.--Aerobiological Decay Rates Of Selected ABOs

Agent	Conditions	Aerobiological Decay Rate (min^{-1})
Francisella tularensis[a]	Day	0.151 at 60% RH 0.254 at 30% RH
Francisella tularensis[a]	Night	0.088 at 60% RH 0.121 at 30% RH
Brucella suis[b]	Night	0.0003 at 85% RH 0.07 at 20% RH
Coxiella burnetii[a]	Day	0.009 at 60% RH 0.040 at 30% RH
Coxiella burnetii[a]	Night	0.009 at any RH
Yersinia pestis[a]	Day	0.492 at 60% RH 0.182 at 30% RH
Yersinia pestis[a]	Night	0.303 at 60% RH 0.111 at 30% RH
Venezuelan Equine Encephalitis[b]	Night	0.02 at 85% RH 0.005 at 20 - 60% RH

Table 8.--Continued

Agent	Conditions	Aerobiological Decay Rate (min^{-1})
Botulinum Toxin[c]	Day	0.037 >50% RH 0.022 <50% RH
Botulinum Toxin[c]	Twilight	0.019 >50% RH 0.009 <50% RH
Botulinum Toxin[c]	Night	0.014 >50% RH 0.007 <50% RH

[a]Source: J. M. Beebe, E. L. Dorsey, N. L. Pollok, E. E. Johns, The Comparative Responses Of *Coxiella burnetii, Pasturella tularensis, Pasturella pestis* And *Serratia marcescens* To Artificial Sunlight At Two Humidities, Technical Memorandum 21, (Fort Detrick, Maryland: U. S. Army Biological Laboratories Fort Detrick, 1962), 1 - 11.
[b]Source: Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, Vol. 2, CB Weapons Today, (New York: Humanities Press, 1973), 128 - 129.
[c]Source: US Army and US Marine Corps, FM 3-3/FMFM 11-17, Chemical And Biological Contamination Avoidance, (Washington, D.C.: Headquarters, Department Of The Army/Commandant Marine Corps, 1992), B-4, B-5.

Figure 1 graphically portrays selected data from Table 8 to give the reader a better appreciation for decay rates. It is interesting to note the significant differences between the bacterium *Francisella tularensis* and the rickettsia-like microbe *Coxiella burnetii*. Also worth noting is the significant effect that sunlight has on both the microbial pathogen *F. tularensis*, and botulinum toxin. An attacker could use these data to tailor the downwind distance (e.g., employ early after sunset to allow maximum downwind travel), and/or persistence in the target area (e.g., use an agent that degrades rapidly in sunlight to allow occupation of the target area within several hours after attack).

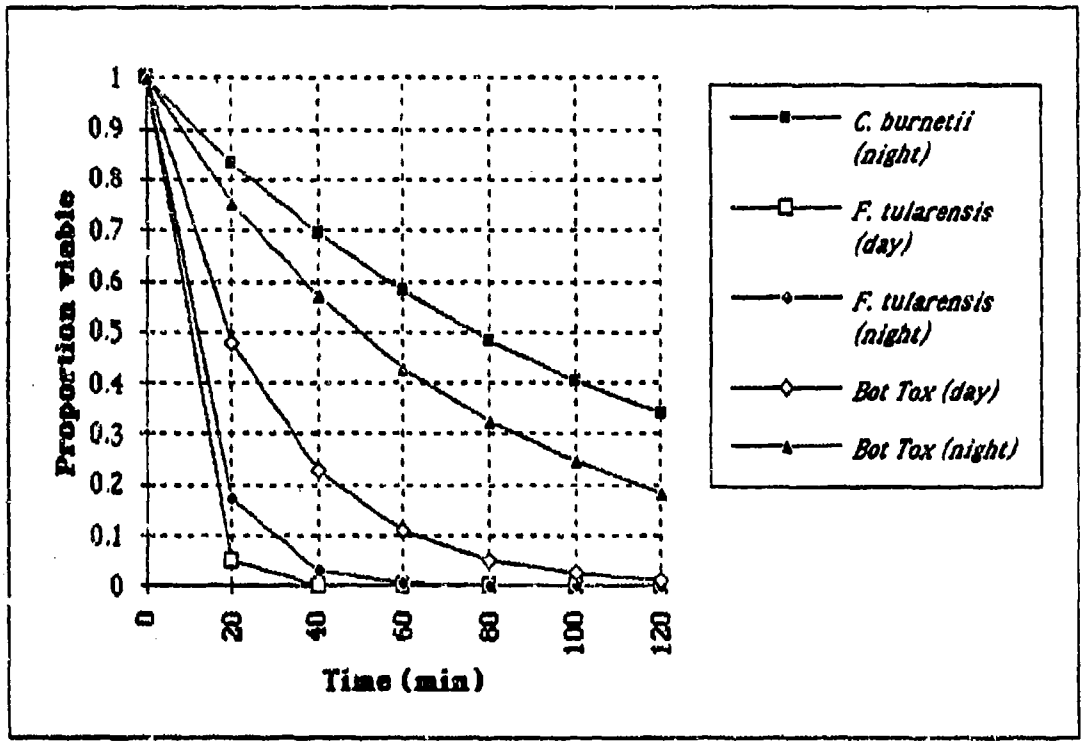

Figure 1. Aerobiological Decay Profiles

13. After delivery, the agent should have a low persistence, surviving only for a short time, thereby allowing a prompt occupation of the attacked area by the aggressor's troops

The factors that affect the persistency of ABOs have already been discussed, as well as the desirability of non-persistency in tactical operations. One factor is the probability of the agent becoming established in a reservoir or creating an epidemic as was discussed in the analysis of criterion 3 (see also Table 4). The other factor is the robustness of the agent in the environment. The same factors listed in the analysis of criterion 12 are relevant here, and so is the discussion of likely attack scenarios.

Perhaps the most persistent of all ABOs is *Bacillus anthracis;* the causative agent of anthrax. In 1942, the United Kingdom performed open-air anthrax tests on Gruinard Island, which lies off the north-west coast of Scotland. As of 1981, the island was still contaminated with viable anthrax spores, and still posted off-limits to

visitors.[41] The only reasonable scenario for an attacker's employment of anthrax on the battlefield would be as part of a "scorched earth" tactic. In this scenario, a belligerent who can only defend is willing to establish a virtually permanently contaminated barrier zone (which would cover a significant area) between his forces and his opponents.

Agents With The Greatest Potential For Tactical Employment

For this section I have taken the results of the previous analyses, and developed a list of ABOs which are most likely to be used at the tactical level. Table 9 represents what I believe are the ABOs best suited for tactical employment. This list does not necessarily conform to threat lists published by other authors. Reasons why this list may differ from others are that I have limited my research to unclassified sources and I have not tried to tailor the list to a particular region. The classified threat lists compiled by intelligence agencies will be more accurate reflections of the ABOs that a particular belligerent is interested in. However, this list is still useful in narrowing the list of all the agents available to a particular belligerent, to those that he is most likely to use on the tactical battlefield. I will discuss differences between this list and a previously published list later.

Table 9.--ABOs Best Suited To Tactical Employment

Agent	Remarks
Incapacitating Agents	
Chikungunya fever	Rapid effects; proven aerosolizable
Dengue fever	Rapid effects; proven aerosolizable
Rift Valley Fever	Rapid effects; no aerosol data available
Venezuelan equine encephalitis	Rapid effects; proven aerosolizable; standardized BW agent
Staphylococcus enterotoxin B	Effective in low concentrations; standardized BW agent

Table 9.--Continued

Agent	Remarks
T-2 toxin	Component of "Yellow Rain"; requires relatively high concentrations to be effective -- a belligerent who has the option of using chemical agents may opt for their use instead
Brucella spp.	Moderate delay for effects (ave. 14 days); standardized BW agent; likely used when battlefield tempo slows
Coccidioides immitis	Moderate delay for effects (7 - 21 days); standardized BW agent; likely used when battlefield tempo slows
Coxiella burnetii	Moderate delay for effects (ave. 19 days); standardized BW agent; likely used when battlefield tempo slows
Lethal Agents (Note: lethal agents are less desirable for tactical employment)	
Francisella tularensis	Mortality rate = 5 - 60%; standardized BW agent; proven aerosolizable; rapid effects
Pseudomonas pseudomallei	Mortality rate up to 100%; proven aerosolizable; rapid effects
Yellow Fever Virus	Mortality rate = 5 - 100%; standardized BW agent; rapid effects
Botulinum toxin	Standardized BW agent; low effective dose; bacterial toxin
Saxitoxin	Effective at low concentrations; bacterial toxin
Diphtheria toxin	Effective at low concentrations; bacterial toxin
Nivalenol	Component of "Yellow Rain"; effective at low concentrations; fungal toxin
Ricin	Used in covert applications; effective at low concentrations; plant toxin
Palytoxin	Effective at low concentrations; may be synthetically produced
Batrachotoxin	Effective at low concentrations; may be synthetically produced
Tetrodotoxin	Effective at low concentrations; may be synthetically produced

To help convey the reasons for the differences between Table 9 and other threat lists, I will use an article that appeared in a recent medical journal.[42] In 1991, the US Army was cited as listing ten ABOs which it considered the most threatening during Operation Desert Storm. The list included: *Bacillus anthracis, Francisella tularensis, Coxiella burnetii,* botulinum toxin, staphylococcus enterotoxin B, Venezuelan equine encephalitis, Rift Valley fever, dengue fever, and hantaan virus. Of these ten agents, there are two that I did not include in Table 9 -- *Bacillus anthracis* (the causative agent for anthrax) and hantaan virus (the causative agent for Korean hemorrhagic fever).

By most criteria, anthrax is a natural choice for tactical employment--it manifests its effects rapidly; it's easy to produce, store and deliver; and it has been a standard BW agent since WW II. But anthrax does possess a couple of serious drawbacks for tactical employment. Pulmonary anthrax (caused by inhalation of aerosolized spores or bacteria) is extremely lethal; which could present a problem when the attacker's forces are in close proximity to the defender's. In the analysis of criterion 13, I mentioned anthrax's extraordinary persistence; which results in large areas of permanently contaminated ground--in effect, a "no-mans land. Of course, the tactical chemical defense officer has to take into consideration the personality of the enemy commander. Leaders like Saddam Hussein, who precipitated the World's worst ecological disaster during the 1990 - 1991 Persian Gulf conflict, would probably show little hesitation in using anthrax.

Hantaan virus is not an ideal tactical agent, but it could be employed in certain situations. The time that it takes to manifest the effects of Korean hemorrhagic fever runs between 12 and 33 days; which means that this agent would best be employed in a relatively static tactical environment. The mortality rate from this disease can run fairly high (1 - 30%), and although there is a vaccine for this disease, its value is questionable. The employer of this pathogen would be wise to release it well away from his own forces.

Alleged But Poorly Characterized Agents

As I stated in Chapter 3, the data that are available regarding these agents are scarce, but I will provide what information is available, along with brief analyses of their applicability to tactical employment. All of these agents are allegedly the products of the recent developments in molecular biology. These deveopments putatively allow for large scale production of novel, or rare, biologically active substances.

Superplague. This agent was allegedly developed by the former Soviet Union, and is a lyophilized, antibiotic resistant strain of *Yersinia pestis*.[43] The technologies required to engineer an antibiotic resistant bacterium and lyophilize it are comparatively routine, so it is not inconceivable that the Soviet Union produced such a strain. However, since it probably is both highly contagious and lethal (presuming little else was done to alter the bacterium's genome), it is not a likely candidate for tactical employment.

Cobra Venom Producing Influenza Virus. According to a 1984 newspaper report, the former Soviet Union was "attempting to inject cobra venom into a common flu virus."[44] While an agent of this sort (highly contagious and lethal) would undeniably have significant psychological impact on the target population, I can not see any reason for expending resources on an agent that has no apparent advantages over naturally occurring pathogens. The rapidity of action associated with straight biotoxin poisoning would be lost, since the influenza virus would first have to reproduce in sufficient numbers to create a lethal toxin concentration within the host. The tactically desirable characteristic of being an incapacitating agent (see table 2) would be lost by the virus due to its expression of a lethal toxin. Because influenza is highly contagious (see table 5) the employer of this agent risks all of the hazards to his own forces presented by contagious/epidemic agents. An additional argument against the use of this particular agent is the possibility that current vaccines will provide defenders immunological protection.[45]

Infectious Nucleic Acids. Nucleic acids are the material that genes are made of, and so are the medium that provides metabolic instructions to cells. It has been known for some time that if a gene can be introduced into a cell intact, it can direct the cell's metabolic machinery to produce the invading gene's product. In fact, there exists a class of pathogens that is known to produce diseases in plants, and is suspected of

causing some animal and human diseases, which consists of free nucleic acid called viroids. The Stockholm International Peace Research Institute (SIPRI) argues that free nucleic acids, which may be derived from existing viral genes, are candidates for ABOs because of their ease of synthesis, infectivity, lack of immunogenicity (i.e., they do not illicit immunological defense mechanisms), and stability in aerosols.[46] While naked nucleic acids are susceptible to enzymatic degradation (nucleic acid-degrading enzymes are ubiquitous), SIPRI noted that they can be protected through combination with basic proteins, lipids, certain polypeptides, and serum albumin proteins.[47] SIPRI also noted that the infectivity of nucleic acids can be enhanced through combination with basic proteins, DMSO (dimethylsulphoxide), polycations and diethylaminoethyl (DEAE) dextran.[48]

While free nucleic acid (or viroid) agents have many characteristics that make them favorable for offensive use, there is one likely characteristic which may argue against their tactical employment. That characteristic is a long period from time of infection to manifestation of disease. My reasoning for this is based on the characteristics of currently known viroid-caused diseases (e.g., scrapie and hepatitis delta agent). These ribonucleic acid-based pathogens are also known as "slow infections."[49]

Psychotropic Substances Or Psychotoxins. These substances have been described as "[m]ind control drugs," which are capable of rendering whole populations incapable of independent thought.[50] Allegedly, the former Warsaw Pact nations had considerable interest in psychotoxins since at least the 1950s.[51] Theoretically, it is possible to isolate a gene (or genes) which control specific mental functions, splice the gene into a rapidly growing microbe, and produce large quantities of psycho-active biochemicals. However, the current state-of-the-art as published in the open literature indicates that large scale production of psychotoxins will not occur for some time. This makes tactical employment of this class of toxins within the next 5 to 10 years highly

unlikely. Manipulation of human genes and gene products is orders of magnitude more difficult than manipulation of bacterial genes.

Gene Altering And Mutagenic Agents. Two mechanisms have been proposed for inducing mutations in target populations' genomes.[52] One proposed mechanism is to use mutagenic chemicals cross-linked to DNA-binding proteins to induce specific mutations. Two problems with this technique include the body's immune system (which would likely recognize the foreign protein and neutralize it), and the intracellular barriers that must be overcome before the agent can reach the targeted genes. Another proposed mechanism is to integrate disease-causing DNA segments into viruses that normally integrate their genes into their host's genes. This mechanism has a better chance of getting the agent into the host's genome, but may still be susceptible to immunological barriers. Both mechanisms potentially have a characteristic which makes them unsuitable for tactical employment--their long time to manifest effects. Because this class of agents depends on manifestation of genetic mutations (e.g., inducement of lymphoma, anemia, or carcinoma), it would probably take weeks to neutralize the target population.

Black Rain Or Blue-X. The former Soviet Union is accused of employing this agent in Afghanistan as a "large population area [incapacitant]."[53] According to the literature, this agent instantly puts people to sleep for two to eight hours.[54] Allegedly, the toxin used in Afghanistan acted so rapidly that intoxicated soldiers were "'frozen' in position before they knew what was happening."[55] However, there is at least one very good argument for questioning the validity of these allegations. Terence White and Kathleen White have stated that ". . .it is inconceivable that an agent can act so quickly that the people 'frozen' in position. . .are not killed by this immediate paralysis of the neuro-muscular system."[56]

Instant Death Or Sleeping Death. The best way to describe this agent is to use a quotation from Anthony H. Cordesman:

> Sleeping death causes instant death to the victim, without affecting the central nervous system. Victims were found in their fighting positions, holding their rifles, their eyes open, their finger on the trigger, and with no apparent cause of death. The agent seems to be odorless and extremely lethal.[57]

The alleged rapid rate of action of this substance would certainly argue for its use as a tactical weapon; but, as mentioned earlier, lethal agents are not particularly well suited to tactical employment. There are a couple of other characteristics not addressed in the available literature that must also be considered in determining its relative merit for tactical use. These characteristics are persistency (short persistency is best), and protection available to the attacking force (e.g., toxoids and therapeutic treatment).

Potential Delivery Means

This section provides two types of information: intelligence indicators, and data for use in downwind hazard predictions. Identifying systems that are capable of delivering biological agents assist the tactical chemical defense officer in his assessment of the potential biological threat. Knowing the capacities of the delivery systems will allow accurate vulnerability assessment of forces, and more accurate prediction of contamination spread after an attack.

Table 10 is a listing of weapons systems that are known or suspected of being capable of biological agent delivery. The ranges and payloads (i.e., maximum weight of agent that may be carried in the missile, rocket, shell, etc.) represent best estimates made by the authors of the open literature sources cited. Classified sources may be able to provide more accurate data. I would like to further caution the reader on a couple of points in interpreting and applying the range and payload data. Biological agent warheads may have significantly different densities than conventional high explosive

warheads. For example, dry preparations of ABOs will have low densities, and so the volume of agent that can fit in the delivery system will likely be the limiting factor before the mass of agent. Also, with bursting munitions a proportion of the agent will be destroyed, or rendered biologically inactive from the low yield explosive used to open the munition and disperse the agent.

The potential also exists for unconventional or "non-military" delivery systems to be used. The technology involved with efficient dispersal of micrometer sized aerosols is also used by farmers and foresters for pesticide application and by paint sprayer designers and heating system engineers for aerosolization of paint and oil.[58] In fact, Theodor Rosebury (who worked in the US biodefense effort during World War II) recalled a lecture he gave to a group of engineers on biological warfare. At the end of the presentation a salesman approached him with an agricultural spray generator brochure, and discussed its potential application to agent dissemination.[59] The possibility of the attacker using such seemingly innocuous systems as farm implements greatly confounds the defender's ability to assess the local threat. What the tactical chemical defense officer should take from this is the fact that an attacker could easily disseminate 500 - 1000 kg "payloads" of agent from a ground-based generator, and so should evaluate his front-line units' vulnerabilities accordingly.

Table 10.--Likely Biological Agent Delivery Systems

System	Range (km)	Payload (kg)	Remarks
Missile Systems			
Condor I	100	unknown	Produced by Argentina; unknown for certain if the warhead can carry ABOs[b, c]
Alacran/Condor II	820 - 980	600 - 1000	Produced by Argentina, Egypt and Iraq; unknown for certain if the warhead can carry ABOs[b, c]
Vector/Condor II	820 - 980	600 - 1000	Produced by Argentina, Egypt and Iraq; unknown for certain if the warhead can carry ABOs[b, c]

Table 10.--Continued

System	Range (km)	Payload (kg)	Remarks
MB/EE-150	150	1100	Produced by Brazil; unknown for certain if capable of carrying ABOs[d]
MB/EE-1000	1000	unknown	Produced by Brazil; unknown for certain if capable of carrying ABOs[d]
MECB VLS	1500	450	Produced by Brazil; unknown for certain if capable of carrying ABOs[d]
SM-70	70	unknown	Produced by Brazil; unknown for certain if capable of carrying ABOs[d]
SS-300	300	2200	Produced by Brazil; unknown for certain if capable of carrying ABOs[d]
Frog-7	65 - 70	455	Possessed by Egypt, Iraq, Iran North Korea, Kuwait, Algeria, Libya, South Yemen, Syria; produced by former USSR; North Korea known to have Chem-Bio warheads[a, b, c, d]
SS-21 Scarab	70 - 120	1318 - 1557	Possessed by Libya, North Yemen, North Yemen, South Yemen, Syria, Iraq; produced by former USSR; Syria and North Korea believed to develop Chem-Bio warheads[a, b, c, d]
SS-23	500	350	Produced by former USSR[b]
Scud-A (SS-1b)	130	900	Produced by former USSR[b]
Scud-B (SS-1c)	300	900	Possessed by Afghanistan, Egypt, Iran, Iraq, Libya, North Korea, South Yemen, Syria; produced by former USSR, North Korea, Egypt; Syria and North Korea known to develop Chem-Bio warheads[a, d]
Scud-C	450	550	Produced by former USSR, North Korea and possibly Syria; Syria known to have Chem-Bio warheads[b, e]
Improved Scud	450 - 600	500	Produced by Egypt and North Korea, unknown for certain if capable of carrying ABOs[b, c]
Scud R-300/R-17E	290 - 320		Possessed by Iran[b]
Al-Hussayn	615	135	Produced by Iraq[a, b, c]
Al-Abbas	920	985	Produced by Iraq[a, b, c]
Tamuz	2000	600 - 1000	Produced by Iraq[a, b, c]
M-9	600	2200	Produced by China, and possibly Syria within the next couple of years[b, e]
M-11	650 - 850	500 - 1000	Produced by China[b]
Prithvi	150	600 - 800	Produced by India; unknown for certain if capable of carrying ABOs[b]

Table 10.--Continued

System	Range (km)	Payload (kg)	Remarks
Agni	1700 - 2400	1000	Produced by India; unknown for certain if capable of carrying ABOs[b]
Hatf I-King Hawk	80	600 - 800	Produced by Pakistan and China; unknown for certain if capable of carrying ABOs[b, c]
Hatf II-King Hawk	300 - 350	800 - 1000	Produced by Pakistan and China; unknown for certain if capable of carrying ABOs[b, c]
Tube Launched Artillery & Rockets			
M-46 130 mm Gun	27.2	74	Produced by former USSR[b]
2S5 152 mm Gun	27	96	Produced by former USSR[b]
M107 175 mm Gun	32.7	147	Produced by the US[b]
BM-21 122mm Multiple Rocket Launcher	20.5	40X17	Produced by former USSR[b]
M-1972 122 mm Multiple Rocket Launcher	20.5	40X?	Produced by former USSR[b]
Cruise Missiles, Remotely Piloted Vehicles, Aircraft-Delivered Bombs & Ground/Vehicle-Based Generators			
SSC-1b 'Sepal' anti-ship missile	unknown	unknown	Hypothesized that this guided missile system could be converted to carry Chem-Bio agents; cited with respect to Syria;[e] may be possible to fit commercially available Global Positioning Systems into otherwise unsophisticated cruise missiles for accurate delivery[i]
DR-3 reconnaissance drone	180	unknown	Produced by Russia; hypothesized may be refitted to carry Chem-Bio agents; cited with respect to Syria[e]
Pchela-1 remotely piloted vehicle	60	unknown	Produced by Russia; hypothesized may be refitted to carry Chem-Bio agents; cited with respect to Syria[e]
USD-2 drone spray system	120	90 liters	A US-developed reconnaissance drone adapted for spraying BW agents[h]
BR-250-WP bomb	depends on aircraft	250	Originally produced by EXPAL of Spain, converted by Iraq to deliver chemical agents[g]
BR-500 HE bomb	depends on aircraft	500	Originally produced by EXPAL of Spain; converted by Iraq to deliver chemical agents, may use both airburst and impact fuzes[f]
Bomb cluster, 750 lb (*Sadeye*)	depends on aircraft	340	Developed by the US in the 1960's for delivery of BW agents[h]

Table 10.--Continued

System	Range (km)	Payload (kg)	Remarks
E41 spray tank, dry agent	depends on aircraft	75 - 140	Developed by the US in 1965 for F100, F105, F-4C and A-4D aircraft[h]
Aero 14B spray tank, liquid agent	depends on aircraft	303 liters	Developed by the US for A-4D, AD-5, AD-6 and FJ-4B aircraft[h]
E22 portable generator		2.6	Developed by the US in the late 1950's for spraying ABOs[h]
E32R1 portable generator		1	Developed by the US in the early 1960s; uses compressed nitrogen to disperse ABOs within 8 seconds[h]

Sources: [a]Harvey J. McGeorge, "Bugs, Gas and Missiles," Defense & Foreign Affairs 18 (May - June 1990), 17 - 19.
[b]Anthony H. Cordesman, Weapons Of Mass Destruction In The Middle East, (London: Brassey's, 1991), 23 - 35, 56 - 57, 72.
[c]Kenneth E. Fess, and Duane E. Williams, Third World Tactical Ballistic Missiles: A Strategy For Defense (U), (Carlisle Barracks, Pennsylvania: US Army War College, 1991), 28.
[d]Joseph S. Bermudez, Jr., "North Korea's Chemical And Biological Warfare Arsenal," Jane's Intelligence Review 5 (January 1993), 226 - 227.
[e]Michael Eisenstadt, "Syria's Strategic Weapons," Jane's Intelligence Review 5 (April 1993), 169 - 173.
[f]Terry J. Gander, "Iraq--Chemical Warfare Potential," Jane's Soviet Intelligence Review 10 (October 1990), 441.
[g]Harvey J. McGeorge, "Iraq's Secret Arsenal," Defense & Foreign Affairs 19 (January - February 1991), 7.
[h]Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical and Biological Warfare, Vol. 2, CB Weapons Today, (New York: Humanities Press, 1973), 82 - 89.
[i]Andrew Mack, More Arms, Less Stability: Nuclear, Chemical, And Missile Proliferation In The Asia-Pacific, (Canberra, Australia: Peace Research Center, Australian National University, 1991), 6.

Analysis Of Biological Attacks' Impacts On Forces

In the previous section I analyzed both the conventional criteria that have been used to evaluate the suitability of biologics for offensive use, and the suitability of putative biological agents for tactical employment. Through that analysis I have developed a list of characterized biologics that are best suited for employment at the

tactical level. To appreciate the actual threat that these agents present, it is necessary to analyze their behavior in the aerosol state. This section analyzes the relationship between particle size and pulmonary deposition, the relationship between particle characteristics and downwind travel, and (using modelling) how these and other characteristics combine to impact on the downwind hazard of aerosolized biologics.

The Respiratory Threat

This section will discuss the interactions of the protective systems of the human respiratory tract with aerosolized particles. First I will review the susceptibility of different regions of the respiratory system to biological agents, then I will discuss the mechanisms of particle deposition, and finally I will discuss the range of particle sizes that pose the greatest threat. This analysis delimits the range of particles used in following analyses.

For purposes of this discussion I will describe the human respiratory system in terms of two distinct regions. The first region includes everything from the nose and mouth to the small, branching ducts of the terminal bronchials in the lungs. This region of the respiratory system protects the more susceptible portions of the respiratory tract by warming and humidifying inhaled air, and by filtering out foreign particles. Particles which impact against the surfaces of this region are trapped in mucous, transported by the ciliated cells lining this region to the esophagus (usually within a matter of hours), and then unconsciously swallowed and gotten rid of.[60] The second region of the respiratory tract is composed of the thin-walled structures from the respiratory bronchioles to the terminating alveolli. This region is only a single cell layer thick and is most susceptible to biological agents for a couple of reasons. First the air-flow within these terminal regions is very low, and this allows time for sedimentation of particles (which is discussed in more detail below). Secondly, because this region functions as the site for gas exchange (oxygen for carbon dioxide),

the surfaces are not covered with protective mucous and ciliated cells. These two characteristics allow microbes and toxins to penetrate the thin membranes and enter the bloodstream.[61]

The mechanisms that play the greatest roles in particle deposition, are inertial impaction, sedimentation, and Brownian motion.[62] Inertial impaction may best be described as the deposition of particles against the sides of the respiratory route due to the combination of the particles' mass and velocity. Heavier particles (~ 7 - 10 μm), rapidly moving in the inspiratory air-flow, have too much inertia to stay with the air currents making rapid turns through the first region described above These particles impact against the protective surfaces and are cleared from the respiratory tract.[63] Sedimentation occurs when the the air-flow slows down or stops (i.e., between inhalations and exhalations), and the mass of the particle is sufficient to allow it to drop onto the walls of the respiratory tract. Sedimentation is the most important mechanism for particle deposition in the sensitive alveolar region.[64] Brownian motion is the chaotic-seeming motion of particles in liquids and gasses, and is most prevalent with particles smaller than 1 μm in diameter. Because of the constant, random collisions that occur between gas molecules and aerosol particles, submicrometer particles (especially those under 0.5 μm) will remain suspended in the gas medium until they randomly impact a surface.[65] The greater proportion of particles with diameters less than 0.5 μm, then, will be removed from the alveolar region during exhalation.[66]

Experimental data have shown that particles within the 0.5 - 5.0 μm range are most likely to be deposited in the susceptible alveolar portion of the respiratory system (2.5 - 3.0 μm diameter particles making up the greatest proportion of deposited particles).[67] It is this particle size range that I will work with for the remainder of the analyses.

One final set of data that is necessary for analysis of the respiratory threat, and modelling of downwind hazards, are air exchange rates. An average person at rest will have an air exchange rate of approximately 0.0007 m^3 at 2 breaths per minute, or 0.0014 m^3/min (1.4 l/min).[68] An average person performing heavy work will have an air exchange rate of 0.0012 m^3 at 36 breaths per minute, or 0.0432 m^3/min (43.2 l/min).[69] A conventional figure which has been used by several workers, and will be used in this thesis in support of hazard modelling, is 0.015 m^3/min (15.0 l/min).[70]

Aerosol Particle Size And Density Relationships To Downwind Travel

The downwind distance that an aerosol particle will travel is inversely related to the particle's settling velocity. A quick review of the formula for settling velocity (V_{TS}) given in Chapter 3 shows that there are two characteristics which affect how fast a particle will drop through the air--diameter and density. The characteristic which has the greatest influence on settling velocity is particle diameter (note that its value is squared). Knowing that the longer it takes a particle to reach the ground from its release point, the farther the particle will be able to travel, it is not surprising that smaller particles travel further downwind. Nor is it surprising that particles with lower densities (e.g., freeze-dried or lyophilized biologics) will travel farther downwind.

Figure 2 provides a graphic comparison of downwind travel distances for particles with diameters in the respiratory threat range, and with densities of 1.0 g/ml (the density of water), and 0.8 g/ml (an arbitrary figure chosen for lack of experimental data on the densities of lyophilized ABOs). Figure 2 neither takes into consideration the filtering effects of vegetation and buildings in the path of the aerosol cloud, nor the loss of biological activity due to aerobiological decay (see Figure 1 above). Downwind travel distances (in kilometers) were calculated using standard temperature and pressure (20° C and 1 atmosphere), a release height of 3 meters, and a

wind speed of 10 kilometers per hour. The reader will note the remarkable differences in potential downwind travel distances between a 0.5 μm diameter particle (about 1000 kilometers), and a 5.0 μm particle (about 13 kilometers).

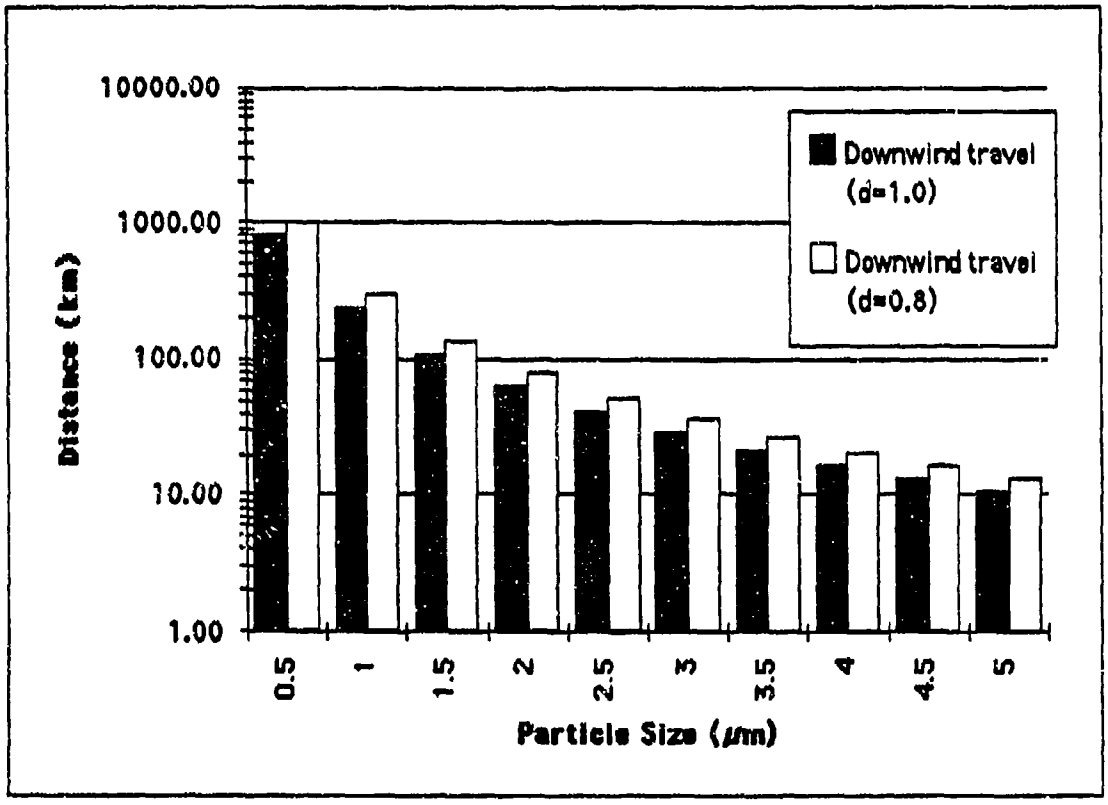

Figure 2. Downwind Travel Distances For Aerosolized Particles

Downwind Hazard Distance Adjusted For Biological Decay

Environmental effects can significantly impact ABOs' downwind hazard distance, and this impact is represented by the aerobiological decay rate. The distances that are represented in Figure 2 are the distances that the particles themselves will travel, but these figures do not necessarily represent how far downwind the biologics will remain biologically active.

Using data from Table 8 (aerobiological decay rates), and the formula for decay of biological agents in Chapter 3, I will illustrate how aerobiological decay can impact downwind hazard distance. In calculating the effects of aerobiological decay, I have made the following assumptions: the mass of agent released = 500 kg (which roughly equates to 1.5×10^{16} organisms for *C. burnetii*, and 1×10^{17} for *F. tularensis*[71]), wind speed = 10 kmph, RH = 60%, release height = 3 m, and the attack begins during darkness with 10 hours before sunrise (worst case--allows use of lowest decay rates for longest duration). In cases where the ABO has relatively low decay rates, and/or different decay rates are available for day and night times, I used a two step process to determine how far downwind a biological hazard would exist. I first calculated the mass or number of microbes still biologically active at the end of 10 hours using the formula for decay of biological agent given in Chapter 3 (D_d). Next, using the mass or number of organisms still surviving at the end of 10 hours in darkness for the starting dose (D), and substituting the particular ABO's LD_{50} number for the value D_d, I solved the equation for time (t):

$$t = \frac{\ln\left(\frac{D_d}{D}\right)}{-k}$$

But for these calculations to be accurate D_d must be corrected for the the factor of particles that are respired into the sensitive regions of the respiratory tract. That is, the value of D_d that defending forces are interested in is the one that will produce an infection or intoxication in unwarned, unprotected persons who may be doing a variety of jobs. For example, the ID_{50} for tularemia is 10 organisms, but for an average person to inspire this many organisms, the agent concentration must be 667 $cfu \cdot min \cdot m^{-3}$. Thus, D_d is adjusted using the formula for total respiratory intake (see Chapter 3) solved for D_d:

$D_d = \frac{I_T}{R}$ where $R = 0.015\ m^3 \cdot min^{-1}$ (an average respiration rate)

I_T = LD/ID_{50} value found in Appendix A.

The above formulae provide the time required (in minutes) for the starting dose to be attenuated to the point where only one effective dose remains. By converting this figure to hours and multiplying it by the windspeed, I am able to calculate the maximum downwind hazard. Of course, this figure will be greater than what would be seen in actual employment, because these calculations do not take dispersion (or dilution) of the ABOs into consideration.

The three ABOs that I have applied to this analysis reveal widely differing downwind hazard distances. The causative agent for tularemia (*F. tularensis*, a non-spore forming bacterium) would only present a hazard for 62 km downwind from the release point. The protein toxin botulinum would have a maximum downwind hazard distance of 180 km. The causative agent for Q-fever (*C. burnetii* which is a fairly hardy organism) could maintain its viability long enough to produce a 610 km downwind hazard.

The next step is to analyze the downwind hazard presented by selected biologics with consideration for dispersion.

Selected Tactical Biological Attack Models

In this section I will use the formulae in Chapter 3 for downwind dosage calculation to analyze the potential hazard of several hypothetical BW attacks. By defining the area that is likely to be contaminated with ABOs in high enough concentrations to [illegible] productive infections or intoxications, one can get a better understanding of the actual vulnerability of the defending force. This same data can be used to determine where to position biological detection units. Timely identification of biological attacks is supported by accurate downwind hazard modelling for at least two reasons. Biological detectors will completely fail to detect the attack if they are outside of the

aerosol plume; so planners need to be aware of the behavior of aerosolized particles under various conditions. Accurate identification of the aerosol (i.e., discriminating between specific ABOs and background detritus) will depend on obtaining samples that have not denatured to the point where identification means are unable to recognize the agent (antibody-based detectors may be especially susceptible to this type of error).

The contour lines delineating the biological hazard area are found by solving the K-theory model, corrected for aerobiological decay, for the crosswind distance value y:

$$y = \sqrt{2\sigma_y^2 \left[-\ln \left(\frac{\left(\frac{D_d}{\exp(-kt)} \right)}{\left(\frac{Q}{2\pi\sigma_y\sigma_z u} \right) \left\{ \exp\left[-\frac{1}{2} \left(\frac{z-H}{\sigma_z} \right)^2 \right] + \exp\left[-\frac{1}{2} \left(\frac{z+H}{\sigma_z} \right)^2 \right] \right\}} \right) \right]}$$

The hypothetical biological attacks that I have modelled represent what I believe are likely scenarios for the tactical battlefield. However, the reader should know that I was restricted in my es due to limitations of data (especially aerobiological decay rates). Four s are modelled below, ranging from use of a non-encapsulated bacterium, to a lethal toxin in an urban environment.

Case A: Tularemia Night Attack

This model is based on a scenario where an attacker fires a Chinese-made M-11 ballistic missile (see Table 10) against a defending force in open terrain. The missile is assumed to deliver 700 kg (1.4×10^{17} organisms) of viable *F. tularensis* at the release point (ground zero), and at a release height of 3 meters. The attack takes place at night, with 10 hours of darkness remaining till sunrise. The windspeed is 10 kmph, the relative humidity is 60%, and the atmospheric stability category is "stable." D_d is corrected for I_τ as described above.

Figure 3 shows the limits of the respiratory hazard generated by the attack. The contour lines represent the distance from the center line of the attack (which is the downwind direction) at which an unprotected person is likely to inhale and retain 10 or more viable organisms (see Appendix A) at 1 meter above the ground.

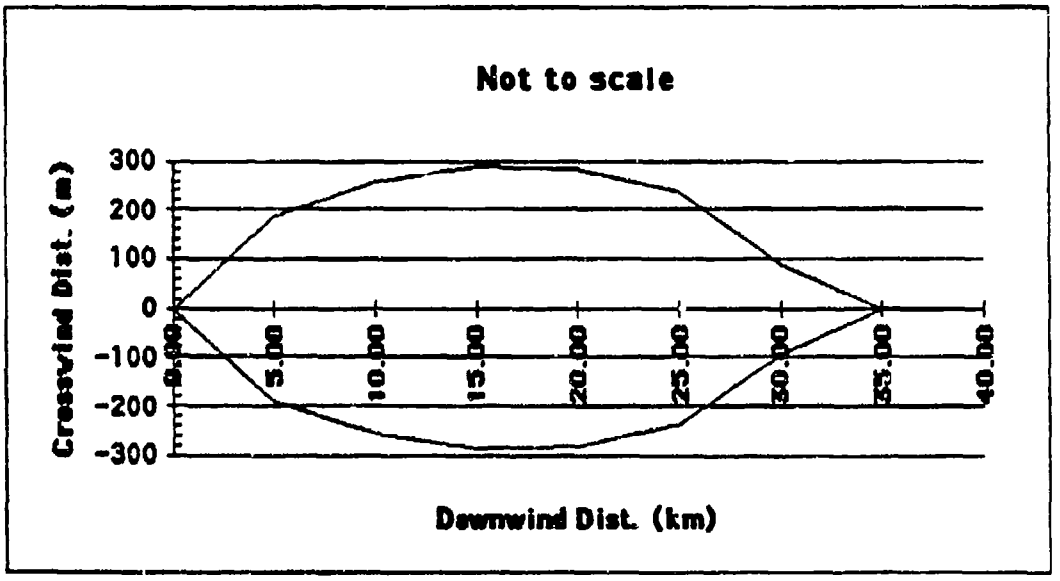

Figure 3. Tularemia Downwind Hazard Profile

For comparison, Figure 4 shows a partially drawn downwind hazard prediction for this same attack using the Simplified Biological Downwind Hazard Prediction (SBDWHP) procedures given in FM 3-3/FMFM 11-17, Chemical And Biological Contamination Avoidance.[72] This figure only plots the dimensions of Zone 1, "The area in which casualties among unprotected troops will be high enough to cause significant disruption, disability, or elimination of unit operations or effectiveness..."[73] The remainder of the plot (Zone 2--"reduced but definable hazard"[74]) would be defined by extending the center line to 320 km, and then drawing a line perpendicular to the center line to intersect the two radial lines. The SBDWHP plot would roughly represent a

triangle with a height and base each measuring 320 km. If Figure 3 was overlaid Figure 4, the boundaries of the threat area predicted in Figure 3 would only extend to 35 km on the center line, and would appear to barely lift above the center line. In other words, the downwind hazard area predicted using the techniques described in Chapter 3 would look more like a sliver in comparison to the fan portrayed by SBDWHP procedures.

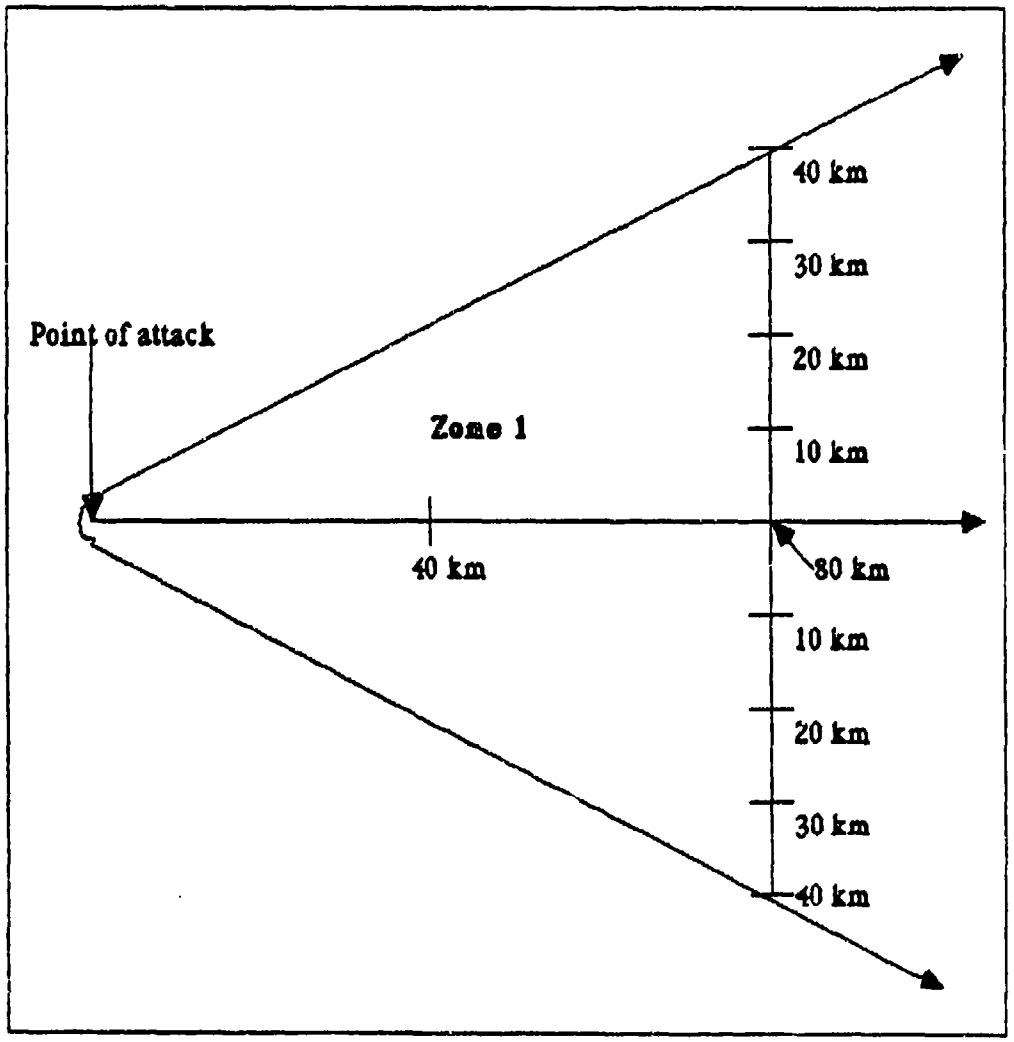

Figure 4. FM 3-3 Hazard Prediction For Case A

F. tularensis represents a class of biological agents which are fairly susceptible to environmental effects, and so it is not too surprising to find such a wide discrepancy between SBDWHP procedures, and those that take into account aerobiological decay. A more hardy agent, such as *C. burnetii*, will have a downwind hazard profile which more closely resembles the plot obtained using SBDWHP procedures.

Case B: Q-Fever Attack

The analyses in the section Downwind Hazard Distance Adjusted for Biological Decay demonstrated that *Coxiella burnetii* (the causative agent for Q-fever) will maintain its viability long enough to present a threat for nearly 3 days. Because of this, the modelling of *C. burnetii's* downwind hazard requires some extra steps. For this scenario, I again used the M-11 missile as the delivery system, and the start of the attack 10 hours before daylight in 10 kmph winds, and with RH = 60%. The next 14 hours (daylight) are characterized by 15 kmph winds and unstable atmospheric conditions, which yield to nightfall, neutral atmospheric conditions and 10 kmph winds. The next day is characterized by slightly unstable atmospheric conditions and 18 kmph winds with 14 hours of daylight, and then neutral conditions and 12 kmph winds during 10 hours of darkness.

Modelling this scenario revealed an important consideration. Attacks that cover more than one set of atmospheric conditions must use average conditions for the mathematical model to work. For atmospheric stability I used neutral conditions, and for the windspeed I used a time-weighted average for the period of concern. For this scenario, which took in 48 hours of changing conditions, the average windspeed is:

$$u = \frac{[(10h \times 10kmph)+(14h \times 15kmph)+(10h \times 10kmph)+(14h \times 18kmph)]}{48h}$$

$$u = 13.8 \text{ kmph}$$

Time-weighted averaging was also used to come up with an average aerobiological decay rate. Figure 5 shows the significant impact that low infectious dose and hardiness have on the downwind hazard area.

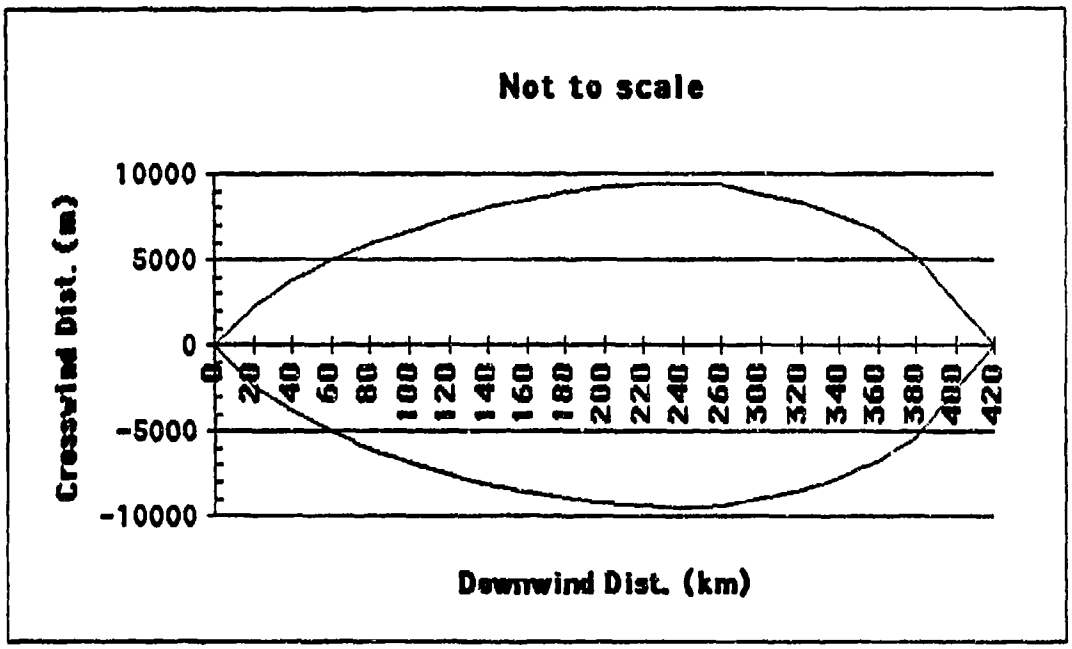

Figure 5. Q-Fever Downwind Hazard Profile

Case C: Botulinum Toxin Attack In Open Terrain

This scenario was used to analyze the impact of a highly lethal protein toxin on the battlefield. In this scenario, the M-11 missile is again used as the delivery system, the target area is open terrain, windspeed for the entire time is 12 kmph, and the RH is 60%. The time of attack has changed to just 5 hours before sunrise. Because botulinum toxin will retain its biological activity long enough to cover varying atmospheric conditions, I used neutral as the atmospheric stability code, and time-weighted averaging to calculate the aerobiological decay rate. The reader will note from Appendix A that there is a range given for the LD_{50} for botulinum toxin. After multiplying the LD_{50} by

the mass of an average person (72 kg), and correcting D_d using the formula for I_T, that range becomes 48 μg at the high dose end (the most toxin it would take to produce a fatality with 50% probability), and 0.15 μg at the low end. Figure 6 is the plot for this scenario. The heavy, dashed line represents the 48 μg contour line; and the light, solid line represents the 15 μg line.

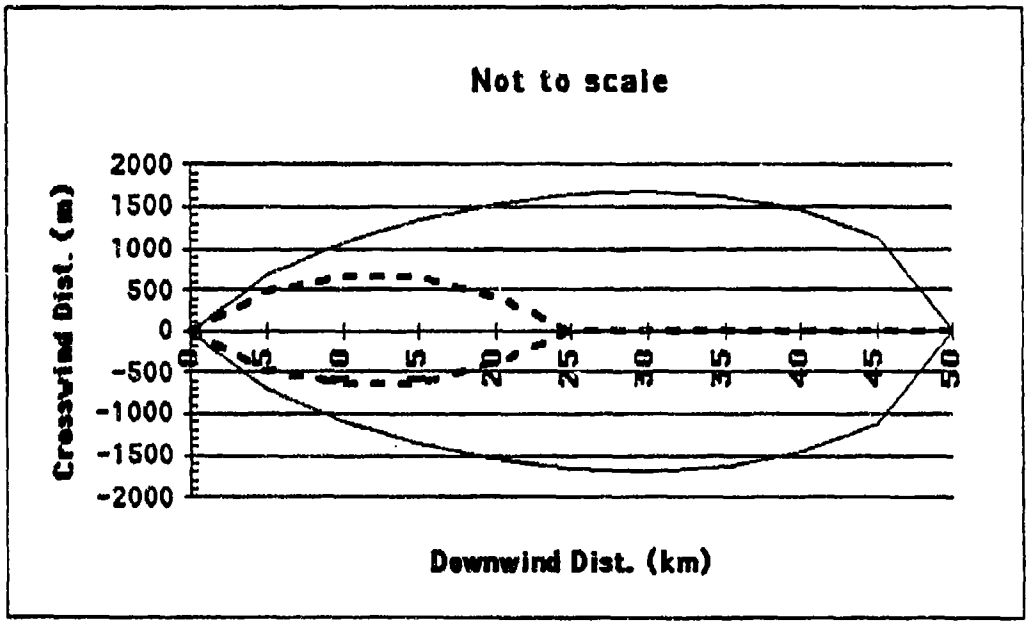

Figure 6. Botulinum Toxin Downwind Hazard In Open Terrain

Case D: Botulinum Toxin Attack In Urban Terrain

This scenario is identical to Case C, except that the attack takes place in urban terrain (see Figure 7). By comparing Figure 7 to Figure 6, one can see the remarkable effect that urban structures have on aerosol migration. Whereas in open terrain this single M-11 missile attack will cover an area nearly 50 kilometers by 34 kilometers, in urban terrain it will only cover an area of about 0.50 kilometers by 0.23 kilometers. As

in Figure 6, the heavy, dashed line represents the 48 μg contour line; and the light, solid line represents the 0.15 μg line.

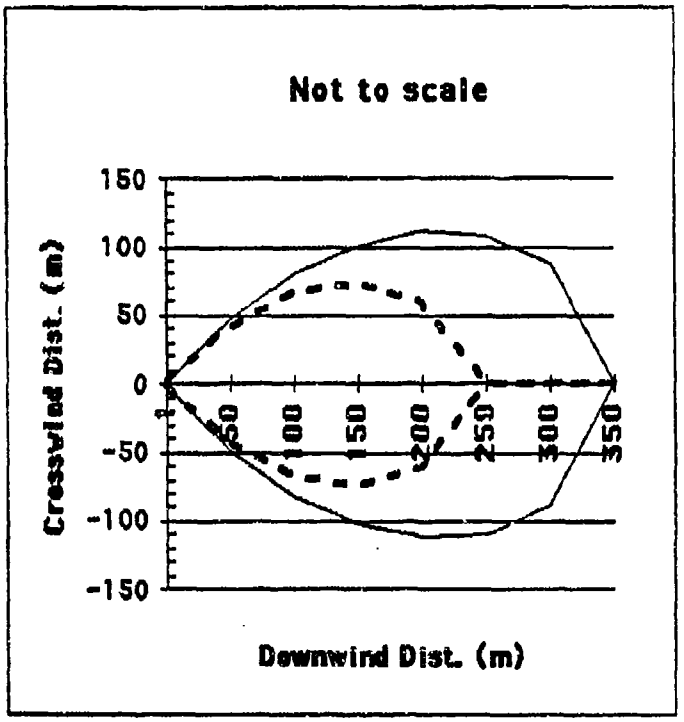

Figure 7. Botulinum Toxin Hazard In Urban Terrain

Other Considerations For Interpreting Downwind Hazard Plots

There are a number of variables that were not included in the downwind hazard plots, but which the tactical chemical defense officer needs to be aware of when predicting biological weapons effects. These considerations include: size distribution of aerosol particles, variable wind direction, and re-aerosolization of particles.

The mathematical model used in this thesis does not take into account the differential fallout of aerosol particles (see Figure 2). Because the larger particles will fallout early (10 - 30 km downwind in a 10 kmph wind), the actual threat of productive infection/intoxication will always be within the contour lines plotted by the model.

The degree to which differential fallout will affect the size of the hazard area will depend on the aerosolization characteristics of the particular weapon system. Some weapon systems may be able to produce aerosols with the majority of particles between 0.5 and 5.0 μm, but I suspect that most (especially bursting munitions) cannot.

The tactical chemical defense officer must be careful to adjust the left and right limits of the contour lines in accordance with changes in wind direction. The mathematical model assumes a single, non-varying wind direction. When the wind direction changes, the chemical defense officer must reorient the downwind direction of the hazard plot to correct for the new direction of cloud travel, similar to the technique described in FM 3-3/FMFM 11-17.[75]

The last consideration for predicting biological hazard areas is the possibility of re-aerosolization of ABOs. This threat is likely to exist predominantly near the point of attack, which is where the larger particles have settled, but are still biologically active. This threat would probably be most prevalent in urban terrain where ABO fallout is in a small area, and where a high volume of traffic is present to carry contamination out of the predicted hazard area. The tactical chemical defense officer must ensure that his forces are warned to stay out of areas that are near the point of attack, and within contaminated urban zones. This warning must remain in effect until after the time biological activity is calculated to be negligible.

Analysis Of Detection Strategies

The purpose of this section is to review current biological identification and detection technologies, and analyze their application to tactical defensive operations. The classes of instruments reviewed range from fairly simple instruments that look only at gross physical characteristics, to those that can identify the particular species of agent(s) in the aerosol.

Aerosol Physical Characterization

Most aerosol characterizing/counting instruments are based on the principle that a particle passing through a beam of light will scatter the light in an amount proportional to the particle's size.[76] Aerosol measurements provide two important pieces of information: aerosol particle concentration, and distribution of particle sizes. Earlier, I stated that particles with diameters between 0.5 and 5.0 μm presented the greatest threat to the respiratory system. With this knowledge, defenders can better estimate the threat that a particular aerosol will present; e.g., the detection of an aerosol predominantly in the 7.0 - 15 μm range presents little respiratory threat, as does one in the 0.01 - 0.1 μm range. An aerosol's distribution profile may also assist in determining the location of agent release in the event that it is unknown. For example, if an average windspeed is 10 kmph, and a release height of 3 meters is assumed, and the distribution profile shows 7 μm particles in the aerosol cloud but no 8 μm particles, the agent release point may be estimated at 4 to 7 km upwind of the sampling point (using the formula for terminal settling velocities given in Chapter 3). By moving the aerosol sizing equipment further upwind, the release point may be more accurately calculated, since the maximum downwind travel distances between the larger particle sizes will narrow. Knowing the time-concentration of particles in the aerosol cloud, or dose (D) in units•min•m^{-3}, will tell the unit whether or not it is likely to suffer casualties. The actual threat can be better assessed if the time of attack is known, so that aerobiological decay can be factored in.

Advantages And Disadvantages. An advantage of these instruments is that they are relatively rapid in producing their data, and can sample the air continuously.[77] Their major disadvantage is that they cannot by themselves discriminate between biological and non-biological aerosols.[78] Thus, dust clouds produced by a convoy of friendly vehicles will be practically indistinguishable from biological attacks.

Immunochemical Techniques

This class of instruments depends on the ability of the immune system-produced proteins called antibodies to identify specific regions on the surfaces of ABOs. Any molecule which can illicit an immunological response can be identified using antibodies. This means that virtually all microbial agents and protein toxins can be identified using this technology. But some non-protein toxins (those that are listed as 'soluble in organic solvents' in Table 13) may not illicit an immune response, and so may not be identifiable with this method. However, antibodies have been produced for the non-protein toxin T-2.

Antibody-tagged molecules are made detectable (either through visual or electronic means) by conjugating enzymes or fluorescent markers to the antibodies. Enzyme conjugated antibodies will produce detectable reactions when specific substrates (i.e., molecules that enzymes specifically interact with) are present. These reactions may result in changes in color or electrical ion concentration. Fluorescent labelled antibodies will emit visible light when exposed to ultraviolet radiation, and the emitted light may then either be visually observed, or electronically quantified.

Antibody-based detection/identification systems can provide two types of information. The most important piece of information is accurate identification of a suspect agent. The remarkable specificity that antibodies have for their target molecule regions (called epitopes) allows them to discriminate between closely related agents, and so reduce the chances of falsely alarming defending forces. For example, antibody based identification systems can distinguish the lethal anthrax bacillus from its benign, but ubiquitous, cousin *B. subtilis*. This class of detectors may also be able to measure agent concentration in terms of mass of agent ($\mu g \cdot min \cdot m^{-3}$ or $mg \cdot min \cdot m^{-3}$).

Advantages And Disadvantages. Perhaps the greatest advantage of antibody-based detection and identification systems is their adaptability. Applications range

from simple, compact, single agent identification systems for use by individual soldiers (e.g., detection "tickets" that turn color when a specific agent is present), to sophisticated, automated systems that are capable of detecting a variety of agents (either in a tactical vehicle mount, or in a clinical laboratory). A disadvantage of antibody-based systems is the relatively long time it takes to get results from a suspect sample (minute to minutes versus the near real time data from aerosol characterizing equipment and mass spectrometers). A potential disadvantage of this class of identification/detection systems is the possibility that novel (e.g., genetically engineered) strains of pathogens may be undetectable by such a specific system. Also, antibody-based detectors may be ineffective at detecting naked nucleic acid agents and viroids.

Biological Activity

This strategy relies on the detection of specific biological processes that are then used to identify individual microbial species. Collins and Lyne's Microbiological Methods, 6th edition, lists 40 biochemical tests that may be used to identify microbes.[79] Unfortunately, the current state of the art of these tests requires culturing the microbes for 5 to 24 hours before readable results can be obtained. But to improve efficiency, there are over two dozen commercially available kits tailored to the identification of specific pathogens.[80]

Advantages And Disadvantages. The principle advantage of these tests is their acceptance throughout the biomedical community. Regardless of what strategies are used to initially alert defending forces to a biological attack, there must be a plan to obtain fresh samples from the attack site for identification using biological activities. Disadvantages of these tests include the time required to obtain results, the need for support equipment (refrigerators, incubators, and other microbiological equipment), and the fact that they are limited to use with bacterial, ricketisial and fungal agents. Toxins and viruses cannot be identified/detected with these tests.

Ion Mobility

This class of detectors works on the principle that particles of differring electrical charge to size ratios will travel at characteristically identifiable speeds in an electric field. Aerosol particles are accelerated to a standard velocity within the instrument, hit with a measured dose of energy to create electrical charges on the particles' surfaces, and then introduced into a chamber where an electrical field exists that tends to retard the charged particles' travel.[81] By calculating the time it takes the particle to travel the depth of the charged chamber, the instrument may be able to identify the particle. Theoretically, each ABO's unique surface composition and size allow these instruments to discern between different biologics. In fact, ion mobility technology is already being used by the Army in the form of the hand-held Chemical Agent Monitor (CAM) to detect mustard and G-nerve agents (tabun, sarin, soman).

Advantages And Disadvantages. A significant advantage of this technology is its near-real time identification, similar to the aerosol physical characterization instrument. Two other advantages of this technology were demonstrated with prototype instruments developed in response to the 1990-1991 Persian Gulf Crisis. The prototypes were capable of employment on aircraft, and were coupled with aerosol counting instrumentation to provide both particle count and composition data.[82] The current disadvantage of this technology is that it is fairly unproven. Until field testing data is collected, the potential for ion mobility detection and identification of ABOs will remain uncertain.

Gas And Liquid Chromatography

Chromatographic techniques rely on the tendency of molecules to differentially fall out of a carrier medium (i.e., an inert gas or liquid solvent) and adhere to a stationary medium based on the molecules' affinity for the stationary medium. The greater a molecule's affinity for the stationary medium, the longer it will take for the

molecule to travel through the separation column. The time a molecule takes to travel through the column (called its residence time) is used to identify the molecule. Combinations of different stationary and carrier media allow for optimization of the process.

Gas and liquid chromatography instruments require the substances of interest be able to travel through the separation columns. For gas chromatography the substance(s) of interest must be volatile enough to enter the gas phase within the instrument's operating temperature range. Liquid chromatography requires that the substance(s) of interest be able to pass through the densely packed separation column. For both techniques microbial agents (and possibly some of the toxins) would have to be broken down prior to analysis. The Stockholm International Peace Research Institute (SIPRI) suggested in 1975 that either a hydrolysis system (to chemically breakup substances) or a pyrolizer (a device that uses heat to breakup large substances) could be fitted upstream of a gas chromatograph to ensure that the instrument could handle the samples.[83] In fact, pyrolizers are currently being used in the prototype Chemical-Biological Mass Spectrometer (CBMS), which will be discussed in more detail later on.

While to my knowledge there are no chromatographic instruments designed specifically for field detection of biological agents, there are several examples of chromatography's potential for this task. One example is the Miniature Continuous Air Monitoring System, or MINICAMS™, which is currently used by Army chemical agent storage facilities. The MINICAMS™ is essentially a miniaturized gas chromatography system that can be configured to operate in a portable mode. Another example of chromatography's potential is the advent of high performance liquid chromatography (HPLC). HPLC allows for rapid, accurate identification and quantitation of substances, and is regularly used in contemporary biology labs.

Advantages And Disadvantages. Two theoretical advantages of gas and liquid chromatography are their potentially great sensitivity and versatility. SIPRI has estimated that gas chromatography's sensitivity could allow detection of as little as 1.5 bacterial cells' worth of microbial products, and its versatility could allow for detection of non-microbial indicators of biological attack such as aerosol stabilizers.[84] Another advantage is chromatography's ability to both identify and quantify substances. One potential disadvantage of chromatography is its complex instrumentation, which may require specialized operator training. The main disadvantage of chromatographic techniques is that they have not yet been evaluated for battlefield detection of biological agents.

Light Detection and Ranging (LIDAR)

LIDAR instruments are stand-off detectors; that is, they may detect aerosols from several kilometers outside of the aerosol plumes. LIDAR accomplishes this through the use of strong pulses of light. When an aerosol cloud crosses the light path, some of the light will be reflected back to the instrument--analogous to the way a radar detects an aircraft using radio waves.[85] Theoretically, ABOs may reflect the light back to the LIDAR instrument at different wavelengths than were used to illuminate the cloud, thus allowing the instrument to both detect the aerosol and identify it as a biological agent.

Advantages And Disadvantages. LIDAR's overwhelming advantage over point detectors (i.e., detectors that must be within the aerosol plume to detect/identify) is that it allows defending forces to identify and react to the biological attack well before the aerosol reaches their positions. Also, the operators are spared the risk of entering the contaminated area. This in turn obviates the logistically burdensome decontamination processes that point detection systems must go through after each attack. A disadvantage of current prototype LIDARs is the light source--a strong laser which has

considerable power requirements, and which is capable of inflicting serious damage on unprotected eyes. As with most of the detection/identification techniques given here, LIDAR's actual ability to identify biological aerosols under field conditions is unknown.

Mass Spectroscopy

This technique uses a particle's profile of mass-to-charge ratios as an identifying "fingerprint." The process requires the original particle to first be broken into smaller, positively charged particles (daughter ions). The ions are then accelerated through a curved magnetic field where they acquire unique trajectories. At the terminus of the magnetic field is a sensor that the particles impact against. The ions' points of impact (or deflections) are recorded and used to calculate their mass-to-charge ratios.[86] Since molecules tend to break up in consistent ways under consistent conditions, their daughter ions' mass-to-charge ratio profiles can be used to identify them. It's this technology that gives the M93 FOX NBC Reconnaissance Vehicle its ability to detect and identify a wide variety of chemical warfare agents.

Up until a few years ago mass spectrometers were limited to identification of relatively small molecules. Now, through the use of pyrolizers, tandem mass spectrometers, and powerful computers, it is possible to obtain useful mass spectrographs of supra-molecular organic substances (up to and including bacteria). A prototype mass spectrometer with these capabilities is the Chemical-Biological Mass Spectrometer (CBMS). The CBMS is currently undergoing testing, and has already demonstrated its potential for battlefield detection of ABOs. If continued development of the CBMS is successful, then it will probably replace the MM-1 mass spectrometer currently on board the M93 FOX NBC Reconnaissance Vehicle.

Advantages And Disadvantages. This technology's greatest potential advantage is its ability to continuously monitor for and identify both chemical and biological warfare agents. Its greatest potential disadvantage could be its inability to identify

biological agents down to the species level. Eventhough the CBMS can take in supra-molecular particles, it still has to break them down into fairly small units for identification, and herein lies the problem. Biological organisms are made up of a very limited number of building blocks; for example, only 20 basic amino acids constitute the bulk of all proteins, and only 5 nucleic acids make up the genes of all organisms (includes uracil in RNA viruses). Biological variation depends to a great extent on the sequencing of these building blocks, and to a lesser extent on unique, small molecular weight products. Of course, many organisms do produce unique, small molecules such as T-2 toxin. The challenge for the CBMS program is to find and exploit the marker molecules that will allow the instrument to distinguish between deadly pathogens and similar, but benign, relatives. Another disadvantage of mass spectrometry is the complexity of the instrumentation. Past experience indicates that these detectors could require significant maintenance support.

Nucleic Acid-Based Techniques

This detection/identification strategy takes advantage of recent developments made in molecular biology. Component techniques of this strategy include automated extraction of genetic material (DNA and RNA), amplification of genetic material (using polymerase chain reaction, or PCR), selective hybridization of nucleic acid sequences, and restriction fragment length polymorphism (RFLP) analysis. These techniques have become standard tools in research laboratories, forensic laboratories, and biomedical industry. Because of the widespread use of these techniques, many of the repetitive, common tasks have been automated; which means that they have become much more "user friendly." Evidence of this strategy's utility can be seen in the rapid, thorough identification and characterization of the causative agent of the Four-Corners Disease, or Hantavirus Pulmonary Syndrome. After the dramatic outbreak of this lethal disease in the Spring of 1993, a cooperative effort was made by several

agencies (to include the US Army) to identify the causative agent. Stuart T. Nichol and co-workers at the Centers for Disease Control (CDC) used PCR techniques to selectively isolate and amplify minute amounts of the pathogen's genetic material from patient tissues.[87] PCR not only allowed this team to confirm the pathogen's relation to other known hantaviruses, but it also provided them with adequate genetic material for further investigation.

I suggest the following possible scheme for employment of genetic detection and identification techniques in a theater of operations. The first step is use of an automated nucleic acid extractor to remove the genetic material of interest from any proteins, lipids, etc. from samples of suspect biological warfare agents. Next, the genetic material in the samples is amplified using non-specific PCR techniques; that is, all of the genetic material is amplified, not just selected pieces. The PCR process also incorporates a label into the copies to allow visualization/detection of the genetic material. The next step uses prepared identification tickets to determine the identity of the unknown genetic material. The tickets have standard nucleic acid sequences already adhered to them, so when the unknown suspension of genetic material is applied to the tickets, only matching sequences adhere (i.e., hybridize to the standards). Finally, the tickets are tested to determine which standard(s) the sample hybridized to. Whichever standard the unknown hybridizes to indicates the identity of the agent.

Advantages And Disadvantages. A significant advantage of this technique is that it allows for detection and identification of naked nucleic acids and viroids. Another advantage is that the sample is being amplified during the analysis (which allows further, more conclusive testing), instead of being destroyed, as in CBMS or HPLC analyses. A potential advantage of a gene-based strategy is that it avoids the problems of antigenic modification of agents; which some believe could defeat antibody-based systems.[88] A disadvantage of this technique is that to my knowledge it has not yet been

packaged and tested for battlefield application. Another disadvantage is that the time it takes to complete the entire process is relatively long (probably more than a couple of hours).

Biological Receptors

Biological receptors are the target molecules that ABOs must interact with to manifest their effects. Examples include the acetylcholine receptors on nerve cells that certain toxins will interact with, and cell surface proteins (docking proteins) that viruses and other intracellular parasites must interact with before they can enter the cell. Generally speaking, receptors are similar to antibodies in that receptors and ABOs must first "recognize" each other before they can interact. However, receptors tend to "recognize" a broader range of agents, and so they may identify a class of agents rather than a specific agent.

The Swedish National Defence Research Establishment (FOA) is currently developing a chemical and biological sensor which uses both antibodies and receptor proteins.[89] The antibodies and receptor proteins are embedded in an artificial cell membrane that is attached to an electronic sensor. The sensor is capable of detecting the conformational changes that occur when a receptor/antibody binds to an agent.

Advantages And Disadvantages. The major advantage of this type of sensor is its ability to detect/identify a broad range of chemical and biological agents. A disadvantage may be the cost, time, and difficulty involved in production of adequate amounts of receptors. Antibodies are produced and excreted in large numbers by immune system cells, but receptor proteins tend to be produced in much more limited numbers--just enough to serve the cell producing them. Another potential disadvantage may be the lack of absolute specificity between receptor proteins and the agents they interact with. It may be necessary to use a complementary identification system that can conclusively identify agents detected by receptor-based sensors.

Ultraviolet/Visible (UV/Vis) Light Spectrophotometry

UV/vis spectrophotometry may be used to identify biochemicals in two ways: (1) through UV light adsorption spectra, or (2) through fluorescence of UV irradiated molecules. UV adsorption profiles for proteins and nucleic acids have been used for years in laboratories for the detection and quantitation of these two classes of molecules. Some biochemicals, such as Aflatoxin B (see Appendix A) and botulinum toxin, will emit visible light under UV irradiation, and so may be detected/identified by characteristic fluorescence spectra.[90]

Advantages And Disadvantages. UV/vis spectrophotometry's greatest advantages are its simplicity and relative speed. The technology used in UV/vis spectrophotometry has been around for years, and so few modifications should be required to develop a field-employable instrument. Using an automated system, an aqueous suspension of suspected ABOs could be analyzed within one to two minutes. A major disadvantage of UV/vis spectrophotometry may be its susceptibility to interference by non-agent, fluorescing particles.[91] The danger of this disadvantage would be an unacceptably high number of false positive alarms.

Summary Of Detection/Identification Strategies

Analysis of candidate detection and identification strategies indicates the strengths and weaknesses of each strategy. However, the analyses also suggest that simultaneous employment of several different detection and identification techniques must be used to correct for weaknesses in any individual technique, and give a more accurate assessment of putative biological attacks.

I mentioned in my analyses those techniques which were developmental, and those that are capable of field employment within a short period of time (several months to a year). I provide some suggestions in Chapter 5 for interim tactical

biological detection/identification until a final,comprehensive biological protection system (and doctrine) is completed.

Endnotes

[1]Erhard Geissler, ed., Biological And Toxin Weapons Today, (New York: Oxford University Press, 1986), 21 - 22.

[2]Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, vol. 2 CB Weapons Today (New York: Humanities Press, 1973), 123 - 124.

[3]Robert Harris and Jeremy Paxman, A Higher Form Of Killing: The Secret Story Of Chemical And Biological Warfare, (New York: Hill and Wang, 1982), 105.

[4]Ibid., 105.

[5]J. W. Cherwonogrodzky and V. L. Di Ninno, Vaccines, Passive Immune Approaches And Treatment Of Biological Agents, Special Publication No. 155, (Ralston Alberta: Defence Research Establishment Suffield, 1992), 11.

[6]Joseph D. Douglass, Jr., "Biochem Warfare: A Warning," Defense Science 2002+ 3 (October, 1987): 66 - 69.

[7]SIPRI, vol. 2, 128.

[8]US Army, US Navy, and US Air Force, FM 3-9, Potential Military Chemical/Biological Agents And Compounds, (Washington, DC: Headquarters, Departments of the Army, Navy and Air Force, 1990), 20.

[9]William C. Hinds, Aerosol Technology: Properties, Behavior, And Measurement Of Airborne Particles, (New York: John Wiley & Sons, 1982), 212

[10]U.S. Army, US Navy, and US Air Force, 56 - 57.

[11]Theodor Rosebury, Peace Or Pestilence: Biological Warfare And How To Avoid It, (New York: Whittlesey House, 1949), 101.

[12]Cherwonogrodzky and Di Ninno, 3.

[13]Ronald M. Atlas, Microbiology: Fundamentals And Applications, 2nd ed. (New York: Macmillan Publishing Company, 1988), 621.

[14]SIPRI, vol. 2, 124.

[15]Atlas, 659 - 660.

[16]Heinz Fraenkel-Conrat, Paul C. Kimball, and Jay A. Levy, Virology, 2nd ed. (Englewood Cliffs, New Jersey: Prentice Hall, 1988), 141.

[17]United Nations Group of Consultant Experts on Chemical and Bacteriological (Biological) Weapons, Chemical And Bacteriological (Biological) Weapons And The Effects Of Their Possible Use, (New York: United Nations, 1969), 16.

[18]US Army, FM 100-5, Operations, (Washington, DC: Headquarters, Department Of The Army, 1993), 1-1.

[19]US Army, FM 100-17, Mobilization, Deployment, Redeployment, Demobilization, (Washington, DC: Headquarters, Department Of The Army, 1992), 1-3.

[20]Geissler, 30.

[21]Cherwonogrodzky and Di Ninno, 3.

[22]Ibid., 12.

[23]Charles Marwick, "Defense Appears To Have Advantage Over Offense Presently In Biological Warfare," Journal Of American Medical Association 265 (13 February 1991), 700.

[24]Ibid.

[25]Marsha F. Goldsmith, "Defensive Biological Warfare Researchers Prepare to Counteract 'Natural' Enemies In Battle, At Home," Journal Of American Medical Association 266 (13 November 1991), 2522.

[26]Ibid.

[27]Geissler, 59.

[28]Peter Williams and David Wallace, Unit 731: Japan's Secret Biological Warfare In World War II, (New York: The Free Press, 1989), 107.

[29]C. H. Collins, Patricia M. Lyne, and J. M. Grange, Collins and Lyne's Microbiological Methods, 6th ed. (Oxford, England: Butterworth-Heinemann, 1989), 52 - 55.

[30]Robin Clarke, The Silent Weapons (New York: David McKay Company, Inc., 1968), 94 - 95.

[31]Geissler, 101.

[32]Ibid.

[33]R. Ian Freshney, Culture Of Animal Cells: A Manual Of Basic Technique, (New York: Alan R. Liss, Inc., 1987), 217.

[34]SIPRI, vol. 2, 69.

[35]Ibid., 64 - 65.

[36]Williams and Wallace, 95 - 97.

[37]SIPRI, vol 2, 312.

[38]Ibid.

[39]Ibid., 64.

[40]Williams and Wallace, 102, 214.

[41]Harris and Paxman, 68 - 73.

[42]Goldsmith, 2522.

[43]John Barry, Daniel Pedersen, and Tom Post, "Planning A Plague?" Newsweek (1 February 1993), 41

[44]Macha Levinson, "Custom-Made Biological Weapons," International Defense Review 19 (November 1986), 1615.

[45]Terence White and Kathleen White, "Biological Weapons: How Big A Threat?" International Defense Review 23 (August 1990), 844.

[46]SIPRI, vol. 2, 319 - 320.

[47]Ibid.

[48]Ibid.

[49]Fraenkel-Conrat, Kimball and Levy, 155 - 157.

[50]Joseph D. Douglass, Jr., "Who's Holding The Psychotoxins And DNA-Altering Compounds?" Armed Forces Journal International 130 (September 1992), 52.

[51]Ibid.

[52]SIPRI, vol. 2, 317 - 319.

[53]Douglass, "Who's Holding The Psychotoxins And DNA-Altering Compounds?" 68.

[54]Joseph D. Douglass, Jr., "The Expanding Threat Of Chemical-Biological Warfare: A Case Of US Tunnel Vision," Strategic Review 14 (Fall 1986), 42, and Anthony

H. Cordesman, Weapons Of Mass Destruction In The Middle East, (London: Brassey's, 1991), 161.

[55]Joseph D. Douglass, Jr., "Soviets Surge In Biochemical Warfare; West Remains Drugged With Apathy," Armed Forces Journal International 126 (August 1988), 58.

[56]White and White, 845 - 846.

[57]Anthony H. Cordesman, Weapons Of Mass Destruction In The Middle East, (London: Brassey's, 1991), 161.

[58]SIPRI, Vol. 2, 72.

[59]Theodor Rosebury, Peace Or Pestilence: Biological Warfare And How To Avoid It, (New York: Whittlesey House, 1949), 103 - 104.

[60]Hinds, 219.

[61]Ibid., 214.

[62]Ibid., 214 - 216.

[63]Ibid., 219.

[64]Ibid., 216.

[65]Ibid., 13, 216.

[66]Hinds, 216, 219; and United Nations Group of Consultant Experts on Chemical and Bacteriological (Biological) Weapons, 61.

[67]Hinds, 220; and United Nations Group of Consultant Experts on Chemical and Bacteriological (Biological) Weapons, 61.

[68]Hinds, 212.

[69]Ibid.

[70]US Army Medical Research And Development Command (USAMRDC), Final Programmatic Environmental Impact Statement: Biological Defense Research Program, (Fort Detrick, Frederick, Maryland: US Army Medical Research And Development Command, 1989), A9-11.

[71]SIPRI, vol 2, 128.

[72]US Army and US Marine Corps, FM 3-3/FMFM 11-17, Chemical And Biological Contamination Avoidance, (Washington, D.C.: Headquarters, Department Of The Army/Commandant Marine Corps, 1992), 4-14 - 4-15.

[73]US Army and US Marine Corps, 4-6.

[74]Ibid.

[75]US Army and US Marine Corps, 3-27 - 3-29.

[76]Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, Vol. 6, Technical Aspects Of Early Warning And Verification, (New York: Humanities Press, 1975), 68.

[77]Ibid., 67 - 68.

[78]Ibid.

[79]Collins, Lyne and Grange, 99 - 107.

[80]Ibid., 97 - 99.

[81]Charles J. Murray, "Detector Identifies Biological Warfare Agents," Design News 48 (20 April 1992), 87 - 88.

[82]Ibid.

[83]SIPRI, Vol. 6, 70.

[84]Ibid.

[85]Ibid., 78.

[86]Robert M. Silverstein, G. Clayton Bassler, and Terence C. Morrill, Spectrometric Identification Of Organic Compounds, 3d ed., (New York: John Wiley & Sons, Inc., 1974), 5 - 6.

[87]Stuart T. Nichol, Christinal F. Spiropoulou, Sergey Morzunov, Pierre E. Rollin, Thomas G. Ksiazek, Heinz Feldmann, Anthony Sanchez, James Childs, Sherif Zaki, and Clarence J. Peters, "Genetic Identification Of A Hantavirus Associated With An Outbread Of Acute Respiratory Illness," Science 262 (5 November 1993), 914 - 917.

[88]Briefing chart by James Campbell, Stephen Walz, Joel Crabble, and Warren Schultz, US Naval Research Laboratory, Washington, D.C.; presented at the "Tri-Service Technology Workshop On Biodetection Systems," (12 - 13 May 1992), US Army Chemical Research, Development and Engineering Center, Aberdeen Proving Ground (Edgewood Area), Maryland.

[89]Ulf Ivarsson, "Tracking Down Poisons," International Defense Review 26 (October, 1993), 823.

[90]SIPRI, Vol 6, 69.

[91]SIPRI, Vol 6, 69.

CHAPTER 5

CONCLUSIONS AND RECOMMENDATIONS

A Summary Of The Threat

The Agents. Chapter 4 presented a number of characteristics which are desirable in ABOs considered for employment at the tactical level. Agents employed at the tactical level would ideally have the following characteristics: (1) they would consistently incapacitate the target population (see Table 2), (2) if toxins are being considered for employment, they should be more effective than corresponding chemical agents (see Table 3), (3) they should not be highly contagious (see Table 4), (4) they should manifest their effects rapidly, although the tempo of operations will determine what is rapid enough (see Table 5), (5) the target population should not have prophylaxes against the ABOs, (6) their use should not be immediately detectable by the target force, (7) the attacking force should have effective prophylaxes against the ABOs, and ideally a way of clandestinely administering prophylaxes, (8) they should be easy and quick to produce, obviating the need for storage facilities (see Table 6), (9) if they must be stored, they should be amenable to lyophilization, (10) they should be capable of aerosol dissemination (see Table 7), (11) their aerobiological decay rates must be known to optimize their employment and to assess their impact, and (12) they should be non-persistent. Known putative agents which best fit these criteria are given in Table 9. Agents that lack sufficient characterization to be evaluated against the criteria are discussed in the section "Alleged But Poorly Characterized Agents."

The Delivery Means. The overriding conclusion from analysis of biological agent delivery means is that there are a remarkable number of ways to deploy tactically significant amounts of ABOs. Table 10 lists forty-one known and suspected biological-capable weapon systems of the "conventional" type; that is, missiles, artillery, aircraft, etc. Many, if not most, of these delivery systems are capable of delivering over 500 kg of agent in a single strike. But just as remarkable are the number and character of unconventional delivery systems. As pointed out in Chapter 4, the wide variety of non-military equipment that could be used for ABO dissemination is potentially great, and certainly enough to confound the tactical chemical defense officer's job of assessing the battlefield threat.

The Potential Impact Of Biological Agents On The Battlefield

The section in Chapter 4 titled "Analysis Of Biological Attack's Impacts On Forces" empirically demonstrates that biological weapons can have significant impacts. A single biological weapon strike is capable of affecting an area tens of kilometers in width, and hundreds of kilometers in depth (see Figures 3, 5, 6 and 7). But the question in Chapter 1 asked "Can biological attacks affect tactical centers of gravity?" My conclusion is yes, biological weapons can significantly affect centers of gravity.

The targets of biological attacks, which may also be tactical centers of gravity, include reserves, logistical support bases, and command and control centers. Reserve forces will typically be positioned well out of range of conventional artillery, but will still be positioned close enough to the main batile area to influence the operation. Operation Desert Storm demonstrated that conventional warhead-tipped ballistic missiles were unable to impact reserves because of their inaccuracy and low yield. But what if those same missiles had been tipped with biological agents? ABO filled missiles would not have to land in close proximity to the reserve formations to inflict significant numbers of casualties. In fact, they would have greater payoffs by landing some

distance upwind to allow their aerosol plumes to spread out and cover the greatest possible area. The potentially high number of casualties within the reserve force would then require logistic support in terms of transportation to medical facilities, consumption of health services resources and possibly decontamination resources, and manpower replacements. The reduced combat effectiveness of the reserves with the concomitant logistical burden reduces the command's freedom of action. The enemy could confound this situation to an even greater extent by employing a lethal toxin against command and control nodes (see Figures 6 and 7). Not only could this defeat the defending force's command and control, but it could also contribute to defeat of national will if the targeted control node is located in an urban area.

Intelligence Indicators

This section's purpose is to provide the tactical Chemical Officer a ready reference for information requirements (IRs) to use in planning. Many of the items given in this section have been discussed in detail in Chapter 4.

The Epidemiological Background Of The Area Of Operations

I. Malek[1] highlights the necessity for knowing what diseases are endemic to an area of operations when he discusses the allegations leveled against the United States by North Korea during the Korean War. In 1952 North Korea persuaded the International Scientific Commission to investigate claims that the United States had attacked several localities with anthrax, plague and cholera infected animals and objects.[2] But among other problems such as forced confessions from American prisoners of war, the lack of epidemiological data on the area frustrated attempts to conclusively determine if the disease outbreaks were natural or the results of biological warfare.

The above paragraph points to two reasons why knowledge of which diseases are endemic to an area of operations is necessary. First, diseases which naturally occur

in a belligerent's area of operations may be more readily exploited. An example of why it is desirable to use "home grown" pathogens may be seen in Iran's attempt to obtain T2-producing fungi from Canada.[3] In this instance the institute which had been solicited was astute enough to be suspicious, and denied Iran's request. Secondly, in the absence of other types of supporting evidence (e.g., unusually high concentrations of aerosolized pathogens) the employer of biological agents may effectively argue that any sudden outbreak of disease is simply due to natural causes, and thus avoid international condemnation for use of biological agents.

The chemical defense officer has several sources available to him for determining which potential ABOs are endemic to a particular area of operations. One is Appendix A of this thesis. Appendix A provides a general picture of particular agents' areas of endemicity. The chemical defense officer should attempt to confirm this information with more detailed research. A useful method is to take advantage of a computerized abstract search service, keying on the particular ABOs of interest and the countries or regions where the unit will be operating. Military medical intelligence channels also keep robust databases on diseases and their areas of endemicity. The chemical defense officer must ensure close coordination with supporting medical agencies early in the planning process.

Socio-Political Factors And Battlefield Dynamics

What is the balance of combat power? Is the enemy likely to use weapons of mass destruction if conditions clearly turn against him? The chemical defense officer will need to continuously update the answer to this question throughout the conflict. The answer also requires some knowledge of the enemy leadership's personality(s)--is the enemy leader the type who disregards international conventions? The reader may want to review Chapter 2 for a more detailed discussion of these factors.

What is the current tempo of operations? Can the enemy reasonably expect 5 to 7 days (or more) of static battlefield dynamics to allow employment of microbial agents? Or is the battlefield so fluid that only toxins or chemical agents can deliver the desired effects in the required time?

Does the enemy possess chemical/biological capable delivery systems (see Table 10)? Friendly forces should be alert for enemy munitions and weapons systems that are capable of chemical/biological delivery.[4]

Has the enemy started psychological operations, or a propaganda campaign, purporting the friendly force's susceptibility to local diseases?[5] These actions could be used to cover biological attacks.

Prophylaxes

Does the friendly force lack effective prophylaxes against agents which may be employed by the enemy? The friendly force's immunization status and access to treatments and chemoprophylaxes should be considered an Essential Element of Friendly Information (EEFI).

What vaccines is the enemy known to possess? A belligerent would be most apt to employ agents that he can protect his own forces from.

Does the enemy have the ability to administer mass vaccinations (i.e., immunize his forces and population) clandestinely? As a minimum, the chemical defense officer should closely monitor reports of immunization programs against unsubstantiated epidemics. Biological detection units should be positioned as close to the enemy's borders as practicable to detect any unusual occurrences of aerosolized biologics which might indicate an aerosol vaccination program in progress.

Do blood samples from enemy prisoners of war indicate that the enemy force has been immunized against ABOs?

Are enemy prisoners of war carrying biological warfare protective items, such as antibiotic tablets, muscle relaxant drugs, and personal immunization records?

Other Intelligence Indicators

Does the enemy have known or suspected ABO production, storage and munitions-filling facilities? The greatest problem in answering this question is the fact that many civilian facilities may be rapidly converted to military use (e.g., pharmaceutical, medical/veterinary research facilities, freeze-drying facilities, and fermented food facilities). However, the sudden appearance of a substantial guard force around these facilities may indicate that they have been converted to military use.[6]

Have sudden and unexplained outbreaks of disease occurred around known or suspected military facilities? The reader may be interested in reviewing allegations surrounding the anthrax outbreak in the Soviet town of Sverdlovsk in 1979.[7]

Does the enemy possess isolated animal research facilities?[8] Isolating biological warfare research and production facilities reduces the chance of a Sverdlovsk-type incident from occurring.

Have unusual amounts of medical research equipment been recovered or found in previously enemy-held territory, that might suggest forward positioned biological warfare labs?[9]

Conditions Supporting Employment Of Biological Weapons

I will address this topic in terms of the influences of socio-political and technological factors, defensive capabilities of the defending forces, battlefield tempo, and environmental factors.

I briefly reviewed the history of biological warfare in Chapter 2, and from that review derived patterns in global and regional developments that supported the use of

biological warfare. I will restate three of those patterns here because of their importance and applicability to current situations. The first pattern, or condition, is technological advance. Historical interest in biological warfare directly paralleled the advances in biology that allowed greater predictability and economy in its use. Significantly, there have been several scientific developments since World War II that support use of biological warfare. Those developments include microbiological culture techniques, aerobiology, immunization technology, synthesis of complex biochemicals, molecular biology and genetic engineering, and advanced intra-theater delivery means. The second condition that supports offensive use of biological agents is international tolerance of unconventional and illegal forms of warfare. International tolerance for use of chemical agents in World War I encouraged Japan's development of a biological warfare program in the 1930's and 1940's. Unfortunately, this same scenario may be playing again because of the international community's failure to take reprisals against Iraq's employment of chemical agents in the 1980's. The final condition that supports employment of biological agents is the perception by one belligerent that its survival is threatened by a greater conventional force. This condition is particularly germane to US contingency operations, since we are practically always going to be able to generate superior conventional combat power in relation to our adversaries.

The defending force's ability to protect itself from biological attack is a critical consideration. The importance of effective prophylaxes was mentioned in several analyses in Chapter 4. The conclusion from these analyses is that a belligerent is most likely to employ biological agents against a defender that neither has effective prophylaxes (immunizations and antibiotics), nor real-time detection/identification capabilities. chemical defense officers must consider their force's immunization and

chemo-phrophylactic statuses when analyzing the threat, and design detection efforts to counter the most likely threats.

Battlefield tempo will determine the type of biological agent used. The enemy will have the greatest flexibility in choice of agent(s) to use during the relatively static lodgement and build-up phase of contingency operations. Active combat operations may limit the enemy's choice, but if he believes he has a reasonable chance of delaying or holding friendly forces for 5 to 7 days, then he can still choose from a number of effective agents (see Table 9). During fast paced operations the enemy will be limited to toxins; they are the only class of biological agents that manifest their effects rapidly enough to affect a dynamic battlefield (see Table 9).

Sunlight, humidity and terrain features have significant impacts on biological weapons' area coverage. Intense sunlight (i.e., clear daylight conditions) may cause a two- to four-fold increase in aerobiological decay rates (see Table 8 and Figure 1). Relative humidity can impact agent survivability to the same degree. Whether the aerobiological decay rates increase or decrease depends on the individual agent--some survive better in high humidity, others in low humidity (see Table 8). While employers of biological agents can partially compensate for aerobiological decay rates by timing their attacks to start soon after sunset, they cannot compensate for the equally important consideration of terrain. Congested terrain such as urban areas may reduce the effective coverage of biological weapons by more than a factor of ten (compare Figures 6 and 7). chemical defense officers must be aware of these environmental factors when performing threat analyses and vulnerability assessments, and when advising their units on the best positions to occupy (dispersed and in built-up or wooded areas).

Battlefield Detection/Identification Of Biological Attack

Conclusive identification of biological attacks is not only necessary for force protection, but it is also critical for the proper conduct of international hearings on uses of weapons of mass destruction. Before conclusive identification of a biological attack can be made, the following types of data must be collected: (1) evidence of unusually high concentrations of aerosolized particles and physical characterization of the aerosol cloud, (2) local meteorological conditions, (3) identification of an agent release point or line, (4) initial identification of the agent(s), and (5) identification of any natural biological aerosol sources. Expeditious collection of accurate data requires biological detection units have the appropriate detection and identification equipment.

The analysis of candidate detection and identification technologies done in Chapter 4 showed that no single technique is capable of obtaining all the required data. Instead, a strategy of integrated technologies must be used. Based on the range of agents which could be employed on the battlefield, and the data requirements outlined above, the strategy must be capable of: (1) collecting physical aerosol data, (2) detecting and identifying both protein and non-protein agents with a high degree of probability, and (3) supporting detailed analysis of putative biological warfare samples using universally accepted methodologies. A suggested strategy is to employ a triad of systems, consisting of currently fielded NBC reconnaissance units using updated software in the M93 FOX Recon Vehicle's MM-1 mass spectrometer (or possibly the CBMS) and antibody ticket detectors; specialized; vehicle-mounted biological detection suites; and battlefield laboratory support teams.

Currently Fielded NBC Reconnaissance Units

Integration of currently fielded NBC reconnaissance teams into the theater biological protection strategy is both necessary and feasible. The number of specialized biological detection systems (such as the BIDS) that will be present in any theater

will probably be insufficient to monitor every potential biological attack site. A reasonable solution is to employ systems that may not be capable of conclusive identification of an attack, but can at least recognize enough indicators of an attack to provide sufficient warning to friendly forces. Specialized biological detection systems could then be directed to the putative attack site for conclusive reconnaissance.

Appendix A and Chapter 4 identify low molecular weight substances that could be used as BW indicators, and which may be detectable by currently fielded MM-1 mass spectrometers. These substances include solvents for non-protein toxins (e.g., ethanol, and dimethylsulfoxide), aerobiological stabilizers for microbes (e.g., glycerol, ethylene glycol, inositol, and glycerol-thiourea), and virus culture medium components (e.g., hydrocortisone). In addition to the MM-1 mass spectrometer, these teams should be equipped with antibody-based ticket detectors to complement MM-1 data. Antibody-based ticket detectors should also be employed to detect BW indicator substances that are too large for the MM-1. Candidates include the serum proteins albumin and transferrin to indicate viral agent attack, protein hormones (e.g., insulin) to also indicate viral agent attack, and the basic proteins called histones to indicate naked nucleic acid agent attack. These measures could probably be implemented in the next one to two years.

Planned future improvements to the M93 FOX Reconnaissance vehicle will greatly enhance its contribution to the overall battlefield detection and identification strategy. The CBMS mass spectrometer, if it proves feasible, will allow current FOX Recon vehicles to become true biological detection systems. Another planned improvement to the FOX is addition of a standoff aerosol detection capability. The current system planned for use is the XM-21 Remote Sensing Chemical Agent Alarm (RSCAAL). It uses infrared sensors to detect aerosol plumes. XM-21 technology is not as capable as LIDAR technology promises to be, but it is a start in the direction of standoff technology.

Specialized Biological Detection/Identification Systems

Specialized, vehicle-mounted, integrated, biological detection systems will provide the backbone of the battlefield biological detection program. At a minimum, this class of systems must be able to collect physical aerosol data, identify agents to the species or specific toxin level, and collect aerosol samples for further analysis.

Physical aerosol data is critical for two reasons: (1) it indicates the concentration of respirable agents in the area, which is necessary to determine if a significant threat exists, and (2) as demonstrated in Chapter 4 this data can be used to estimate the location of agent release. Superimposed on this data must be the date, time and location that it was collected. This additional data will allow follow-on analyses to determine if the presence of the agent aerosol was due to BW attack, or downwind effluent from a natural biological aerosol generator.

The method used to specifically identify the agent must be both rapid and reliable. The need for reliability should be met through a combination of proven and complementary technologies. Based on the analyses of detection systems in Chapter 4, and the use of complementary detection and identification strategies, I propose that the following technologies be integrated into the system. First, antibody based detectors should be used for their proven ability to specifically identify supra-molecular biological substances (up to and including microbes). Second, either a high performance liquid chromatography (HPLC) system or gas chromatography system, with upstream hydrolyzers or pyrolizers, should be included to detect high molecular weight agent markers, and to provide characterization data for novel agents that can not be detected by the antibody-based detector. Finally, integrated with the aerosol counter, there should be either an ion mobility-based detector or an automated ultraviolet-visible light (UV/vis) spectrophotometry system for immediate, albeit non-specific, detection of BW agents.

As good as this system may be at detecting, rapidly identifying, and quantifying the aerobiological threat, there must still be a system for collecting raw samples for further analyses. In the event that an enemy should employ biological agents, our national authorities, and probably the international community, will certainly be interested in closely analyzing all data collected on the battlefield. The best way to ensure credibility is to back up the data obtained from the novel, automated detection techniques with conventionally accepted laboratory techniques. For this reason, there must be a module within the suite that synchronously collects samples in a physiological solution (i.e, the same acidity, salinity, etc. as human cells or blood plasma) as the automated system analyzes the aerosol particles.

Supporting Battlefield Laboratories

This part of the biological detection and identification triad will perform its duty after initial protective measures have been implemented. However, it is no less important than either of the other two parts of the triad. I recommend two identification strategies for employment at this level: biological activity-based identification techniques, and nucleic-acid based identification techniques. Biological activity tests have long been the standard in the biomedical community for the identification of pathogenic micro-organisms, and so should be used whenever possible. Nucleic acid-based identification techniques, as typified by the PCR techniques discussed in Chapter 4, can both confirm results from other tests, and identify some agents not detectable by biological activity. This technology can be used to detect viruses, naked nucleic acid agents, and possibly microbes that have lost their vitality due to environmental exposure or improper handling during sampling and transport. Samples which yield negative results to these two tests could be toxins. Suspected toxins should be sent to a laboratory capable of running detailed and sensitive immunochemical, chromatographic and live animal tests.

Critical Tasks For Biological Detection Units

The analyses and conclusions presented to this point suggest a number of tasks that must be successfully executed to ensure force protection. A number of those tasks are directly related to the planning and execution of theater biological detection plans, and so are the tasks that biological detection units must be proficient in to fulfill their mission. I propose the following critical tasks (i.e., mission essential tasks) for biological detection units.

Assessing The Biological Threat In The Area Of Operations (AO)

Determine which agents are endemic to the area. Medical intelligence agencies will have data on disease occurrences for the region that the AO is a part of. Therefore, early and continuous coordination with supporting medical units is critical. Medical journal abstract searches can provide valuable data for use during predeployment planning. Finally, Appendix A may be used as a general guide for pathogen endemicity.

Determine which agents are most likely to be used on the tactical battlefield. Classified reports of the enemy's capabilities and known or suspected agent arsenals should be compared to the list of agents in Table 9 to refine the tactical threat list. The most dangerous threat agents are those for which we possess neither vaccines nor chemoprophylaxes. The agents that the enemy is most likely to employ are those he possesses prophylaxes for. Those agents that appear on both lists present the greatest threat to our forces.

Identify potential delivery means. Using enemy order of battle data (developed by supporting intelligence agencies) and information in Table 10, identify probable biological agent delivery systems. This information will provide the range and payload data necessary for conducting vulnerability analyses.

Assess the impact of current and projected battlefield dynamics on the enemy's decision to employ ABOs. Assess the vulnerability of debarking units in ports--how concentrated are the forces, how long are forces remaining in the lodgement areas? Determine how closely matched the opposing forces are--does the enemy leadership have reason to believe that its survival is at stake? Is the campaign moving so rapidly that microbial agents would be ineffective, or is the battlefield fairly static?

Detection, Identification And Alerting Of Supported Forces

Establish a system for the rapid, controlled evacuation of attack site samples to supporting laboratories. The system should be formalized (e.g., as a part of plans and orders, or memoranda of agreement), and exercised prior to actual need. There must be a means for evacuation of samples to a responsible laboratory in the US. Examples are the US Army Medical Research Institute For Infectious Diseases at Ft. Detrick, Maryland; the US Army Chemical Biological Defense Command at Aberdeen Proving Ground, Maryland; or the Life Sciences Division at US Army Dugway Proving Ground, Utah. Since preservation of the samples is absolutely necessary, some method of keeping the samples at about 4° C during transport must be available.

Position biological detectors to cover high value targets that are likely to be targeted by the enemy (e.g., command, control and communications nodes; troop concentrations; airports and seaports; critical weapons systems). The detection and identification plan must provide for 24 hour operation of the biological detection systems. Provisions must be made for radio-telephone links with both adjacent units and higher headquarters. This will require coordination with signal units for proper Signal Operating Instructions (SOIs, a.k.a. CEOIs). Whenever possible, avoid positioning biological detectors downwind of natural biological aerosol generators, such as animal and food processing plants, pharmaceutical plants, ranches, sewage treatment plants, and landfills.

Detect and make preliminary identification of ABOs within 10 - 15 minutes of agent aerosol cloud arrival.

Within 5 minutes of detection and identification of a probable biological attack, alert adjacent and supported units.

Within 45 minutes after the initial alert complete a downwind hazard assessment using automated techniques (see the modelling techniques in Chapter 4), and transmit a refined NBC downwind hazard report.

Convey raw samples and all relevant data to supporting laboratories and intelligence agencies for detailed analyses. Along with the agent samples the following sets of data must also be transmitted: physical aerosol data, initial identification data, local meteorological conditions, any possible natural sources of biological aerosols, sample identification data (i.e., an ID number), and chain of custody records.

In Conclusion: A Design For Tactical Biological Defense

If the sum of this thesis were to be distilled into a few fundamental points, they would be the following. First, the potential for biological attack against US forces is greater today than at any time in modern history. Second, the variety of biological agents and agent delivery systems make careful, thorough intelligence preparation of the battlefield a necessity. Third, mathematical tools can be used to accurately assess our forces' vulnerabilities, to analyze actual biological attacks for downwind hazards, and to estimate delivery locations and methods. Fourth, accurate, rapid identification of biological attacks is critical to force protection. And finally, accurate and rapid BW attack identification requires the complementary employment of multiple sensor technologies, and the necessary skill to integrate and interpret data from the sensor/detector instruments.

Recommendations For Further Study

One of my objectives in writing this thesis was to provide a consolidated "baseline" of tactical biological defense information. The purpose of this baseline is to provide a starting point for other, more specific inquiries, and to stimulate development of novel strategies for biological defense. In collating the data that supports this thesis, I have found many omissions in certain classes of data. In various analyses I have found requirements for further research that is necessary for a truly integrated and flexible biological defense program. I will list the areas that could significantly benefit from future research.

Accurate downwind hazard predictions require accurate agent data. The reader will note that many of the ID_{50}/LD_{50} entries in Appendix A are blank. These data should be made available to chemical defense officers, as well as aerobiological decay data for the 60 ABOs that are not listed in Table 8. Other classes of data necessary for accurate downwind hazard prediction include aerosol particle size distribution profiles produced by various delivery means, typical release heights of the different delivery means, and densities of aerosolized biological particles.

The mathematical model used in this thesis is very useful, but there is a need for further refinement. Specifically needed are diffusion parameters (see Table 1) for different types of terrain. For example, information on tropical jungle, temperate forest, brush and scrub-covered plains should be provided. It is preferable to have the atmospheric stability categories in Table 1 matched with the 7 atmospheric stability categories in FM 3-3, Chemical and Biological Contamination Avoidance.[10]

My analyses of potential detection and identification strategies and technologies are based on fairly "generic" laboratory-oriented literature. Testing of field variants of these instruments under simulated battlefield environments is necessary for accurate evaluation of their capabilities.

Endnotes

[1]I. Malek, Biological Weapons, edited by Steven Rose, CBW: Chemical And Biological Warfare (Boston: Beacon Press, 1968), 48 - 61.

[2]Robin Clarke, The Silent Weapons, (New York: The David McKay Company, Inc., 1968), 22 - 26.

[3]Barend ter Haar, The Future Of Biological Weapons (New York: Praeger, 1991), 52 - 53.

[4]Peter Williams and David Wallace, Unit 731: Japan's Secret Biological Warfare In World War II, (New York: The Free Press, 1989), 103.

[5]Ibid., 108

[6]Ibid.

[7]Elisa D. Harris, "Sverdlovsk And Yellow Rain: Two Cases Of Soviet Noncompliance?" International Security 11 (Spring 1987), 41 - 95.

[8]Williams and Wallace, 103.

[9]Ibid., 106, 110.

[10]US Army and US Marine Corps, FM 3-3/FMFM 11-17, Chemical And Biological Contamination Avoidance, (Washington, DC: Headquarters, Department Of The Army/Commandant Marine Corps, 1992), 3-9.

GLOSSARY

Agent(s) of Biological Origin (ABO(s)). Any organism, toxin, or chemical derived from a biological system/organism, to include: microbial pathogens, toxins, physiological regulators and other biochemicals.

AO. Area of operations.

Biological Detection. Refers to being made aware of an unusually high concentration of ABOs in the environment, which may or may not be due to offensive biological operations.

BIDS. Biological Integrated Detection System--the US Army's proposed truck-mounted suite of biological agent detection and identification systems tentatively scheduled for fielding around 1996 - 1997.

Biological Protection. Refers to all measures, active and passive, that are taken to minimize the effects of an enemy's employment of biological weapons against friendly forces.

BW. Biological warfare.

C. Centigrade.

CBMS. Chemical-Biological Mass Spectrometer--the current concept is for a suite of biological and particle detection/quantitation instruments to be carried on a high mobility multi-purpose wheeled vehicle (HMMWV). Particle sizing/counting, and anti-body based detection/identification technologies are two of the central technologies being considered for employment in the system.

C_C. Cunningham correction factor (no dimensions).

cfu. Colony forming units--the number of microbes that can replicate to form visible growth on a solid growth medium, as determined by the number of colonies that develop.[1] A colony may be formed by one or more individual microbes, and is considered the standard means for quantifying microbial numbers.

Conclusive Identification Of A Biological Attack. Means that enough data has been collected to conclude that offensive employment of ABOs has been made by a belligerent--the available evidence will stand-up in an international court of law

cm. Centimeter(s).

d. Particle diameter.

D_d. The biologically active (viable) dose in units•minute•m^{-3}.

DNA. Deoxyribonucleic acid -- the material that makes up genetic material in most microbes, the exceptions being RNA viruses and viroids.

η. Viscosity of air, which at 20° C is 1.81×10^{-4}.

Effective dose. The dose of a chemical or biological agent which is required to manifest its effects (e.g., death, incapacitation).

exp. The exponential function e^x, where $e \approx 2.71828$.

g. Acceleration of gravity, which at sea-level is 980 cm/sec.

GC. Gas chromatography/gas chromatograph.

gm. Gram(s).

h. Hour or hours.

H. The height of burst or release height of the aerosol generator (in meters).

HPLC. High performance liquid chromatography/high performance liquid chromatograph.

Identification of ABOs. Refers to conclusive determination of what the ABO is (e.g., species of a pathogen, identity of a toxin).

Incapacitating Agent(s). Refers to those agents of biological origin which are capable of rendering persons incapable of performing their normal duties[2] and which have a typically low mortality rate.[3]

ID_{50}. Infectious Dose 50; the number of microbes required to effect a productive infection in a person with 50% probability, or Incapacitating Dose 50; the dose required to incapacitate a person with 50% probability.

k. The aerobiological decay rate (in per minute, or min^{-1}).

km. Kilometer(s).

kmph. Kilometers per hour.

l. Liters.

λ. Mean free path for air, which at 1 atmosphere and 20° C is 0.066 μm.

LD_{50}. Lethal Dose 50; the dose required to effect death in a person with a probability of 50%.

LIDAR. Light detection and ranging.

ln. The natural logarithm function using the base e (≈ 2.71828).

m. Meter(s).

μm. Micrometer(s).

min. Minute(s).

MS. Mass spectroscopy/mass spectrometer.

NBC. Nuclear, biological and chemical.

pfu. Plaque forming units--the number of viruses that can replicate to form clear patches (plaques) on a continues sheet of host cells in growth medium, as determined by the number of plaques that develop. A plaque may be formed by one or more individual viruses, and is considered the standard means for quantifying viruses.

π. The constant 3.1415927.

PCR. Polymerase chain reaction--a method of replicating a few copies of a gene (or genetic material) into a large number of copies to facilitate further manipulation and investigation.

Q. The source strength of the biological aerosol generator expressed in appropriate units (cfu, pfu, or μg).

R. Respiratory minute volume in $m^3 \cdot min^{-1}$.

RFLP. Restriction fragment length polymorphism--a phenomenon that results in identifiably different patterns when DNA or RNA is treated by enzymes which cut the nucleic acid sequence at specific sites (restricts the nucleic acid); different sequences result in restricted segments of different lengths, and the segments are seperated and identified on the basis of their length.

RH. Relative humidity (%).

RNA. Ribonucleic acid--a class of materials that perform "messenger" duties between genes and the protein making apparatuses of a cell, and may act as enzymes. In other organisms (i.e., RNA viruses and viroids) it is the genetic material.

ρ_p. Density of the material being aerosolized in grams per milliliter (g/ml)

sec. Second(s).

σ_y. The standard deviation of concentration distribution in y (crosswind) direction (see Chapter Three for calculation).

σ_z. The standard deviation of concentration distribution in z (vertical) direction (see Chapter Three for calculation).

t. Time of travel given in downwind distance traveled (m) divided by the wind speed in minutes (min).

Tactical-Level Operations. Refers to those battlefield operations conducted by corps and below, both during war and operations other than war.

Toxoid. A modified toxin of biological origin which will illicit an immunological reaction, but which has lost its toxicity.[4]

u. The variable wind speed in x (down-wind) direction.

Vaccine. A preparation used to stimulate a protective immunological response in the recipient (vaccinee).

V_{TS}. Settling velocity for aerosolized particles in centimeters per second (cm/s).

y. Distance in cross-wind direction (in meters).

z. Distance in vertical direction (in meters).

Endnotes

[1]Ronald M. Atlas, Microbiology: Fundamentals And Applications, 2nd ed. (New York: Macmillan Publishing Company, 1988), 747.

[2]Erhard Geissler, Biological And Toxin Weapons Today, (New York: Oxford University Press, 1986), 171.

[3]Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, Vol. 1, CB Weapons Today, (New York: Humanities Press, 1971), 123.

[4]Atlas, 778.

ANNEX A

TABLES OF POTENTIAL AGENTS OF BIOLOGICAL ORIGIN

The following tables represent a compendium of potential biological warfare agents, and selected characteristics. Data presented in the tables are extracted from:

W. C. Anderson III, and J. M. King, Vaccine And Antitoxin Availability For Defense Against Biological Warfare Threat Agents, (Fort Sam Houston, Texas: U.S. Army Health Care Studies and Clinical Investigation Activity, U.S. Army Health Services Command, 1983), 1 - 36.

Ronald M. Atlas, Microbiology: Fundamentals And Applications, 2nd ed., (New York: MacMillan Publishing Company, 1988),618, 642, 649, 655 - 713.

Susan Budavari, Maryadele J. O'Neil, Ann Smith, Patricia E. Heckelman, The Merck Index: An Encyclopedia Of Chemicals, Drugs, And Biologicals, 11th ed., (Rahway, N.J.: Merck & Co., Inc., 1989), 2, 30, 157, 226 - 227, 383, 407, 1052, 1107, 1307, 1330, 1541.

Robin Clarke, The Silent Weapons, (New York: David McKay Company, Inc., 1968), 250-253.

J. W. Cherwonogrodzky and V.L. Di Ninno's, Vaccines, Passive Immune Approaches And Treatment Of Biological Agents, (Ralston, Alberta: Defence Research Establishment, Suffeild, May 1990), 1-33.

James A. F. Compton, Military Chemical And Biological Agents: Chemical And Toxicological Properties, (Caldwell, New Jersey: The Telford Press, 1987), 336 - 408a.

Heinz Fraenkel-Conrat, Paul C. Kimball and Jay A. Levy, Virology 2nd ed., (Englewood Cliffs, New Jersey: Prentice Hall, 1988), 96-412.

Erhard Geissler, Biological And Toxin Weapons Today, (New York: Oxford University Press, 1986), 24, 36-41, 44, 58, 61.

James M. Hughes, C. J. Peters, Mitchell L. Cohen, and Brian W. J. Mahy, "Hantavirus Pulmonary Syndrome: An Emerging Infectious Disease," Science 262 (5 November 1993): 850 - 851.

Eliot Marshall, "Hantavirus Outbreak Yields To PCR," Science 262 (5 November 1993): 832 - 836.

Stuart T. Nichol, Christina F. Spiropoulou, Sergey Morzunov, Pierre E. Rollin, Thomas G. Ksiazek, Heinz Feldmann, Anthony Sanchez, James Childs, Sherif Zaki, Clarence J. Peters, "Genetic Identification Of A Hantavirus Associated with An Outbreak Of Acute Respiratory Illness," Science 262 (5 November 1993): 914 - 917.

Stockholm International Peace Research Institute (SIPRI), The Problem Of Chemical And Biological Warfare, vol. 2, CB Weapons Today (New York: Humanities Press, 1973), 38 - 39, 42, 102 - 103, 246.

The United Nations Group of Consultant Experts on Chemical and Bacteriological (Biological) Weapons, Chemical And Bacteriological (Biological) Weapons And The Effects Of Their Possible Use, (New York: United Nations, 1969), 92-93.

U.S. Army, US Navy and US Air Force, FM 3-9, NAVFAC P-467, AFR 355-7, Potential Military Chemical/Biological Agents And Compounds (Washington: Headquarters, Departments of the Army, Navy, and Air Force, 1990), 100-105.

US Army and US Marine Corps, FM 3-3, FMFM 11-17, Chemical And Biological Contamination Avoidance (Washington, D.C.: Government Printing Office, 16 November 1992), B-0 to B-5.

Table 11.-- Bacteria, rickettsia, and fungi

Causative Agent	Disease	Latent Period (Days)	Vaccine Y/N/D[1]	Mortality (%)	Infectious Dose 50% (ID_{50})	Treatment	Areas of endemicity
Bacillus anthracis	Anthrax	1-4	Y	25-100	1300 spores	penicillin ciprofloxacin doxycycline	Worldwide
Brucella spp	Brucellosis	6-60 (ave-14)	D	2-6	~10	doxycycline tetracycline streptomycin	Worldwide
Chlamydia psittaci	Parrot fever or ornithosis	1-4		10-100		tetracyclines	tropical Central & South America
Coccidioides immitis	Coccid-ioidomycosis	7-21	N	0-50	1359 spores	Amphotericin B, chloramphenicol	Arid regions of the Americas & former USSR
Coxiella burnetii	Q Fever (Query fever)	10-26 (ave-19)	Y	1-4	1-10	tetracycline chloramphenicol	Worldwide, except Iceland & Scandinavia
Francisella tularensis	Tularemia	1-10 (ave-3)	Y	5-60	10	streptomycin gentamicin doxycycline	North America, Japan & parts of former USSR
Legionella pneumophila	Legionnaires Disease[2]			15-17		erythromycin, tetracycline	First diagnosed in Philadelphia, U.S.
Pseudomonas mallei	Glanders	2-14	N	up to 100	3200	sulphadiazine, chloramphenicol, tetracycline	Europe & Asia
Pseudomonas pseudomallei	Melioidosis	1-5	N	up to 100		A tetracycline, chloramphenicol	Far East
Rickettsia prowazekii	Infectious or classic typhus fever	5-23 ave - 11	Y	10-50		chloramphenicol, tetracycline, doxycycline	3d world areas in Africa, Asia & Latin America

Table 11.--Continued

Causative Agent	Disease	Latent Period (Days)	Vaccine Y/N/D[1]	Mortality (%)	Infectious Dose 50% (ID_{50})	Treatment	Areas of endemicity
Rickettsia rickettsii	Rocky mountain spotted fever	2-14 ave - 7	Y	10-30		chloramphenicol, tetracycline	North & South America; usually in Spring & Summer
Rickettsia tsutsuga-mushi	Scrub typhus	6-19				chloramphenicol, A tetracycline	Eastern & Southeastern Asia
Rickettsia typhi	Murine or endemic typhus fever	6-14		2		chloramphenicol, A tetracycline	Worldwide; peaks in Summer
Salmonella typhi	Typhoid Fever	7-21	Y	up to 20	10-50	chloramphencicol, tetracyclines, streptomycin, kanamycin	Worldwide
Shigella spp.	Dysentery	1-3	N	2-20	5000 (oral)	tetracycline, ampicillin, nalidixic acid	Worldwide
Vibrio cholerae	Cholera	1-5	Y	up to 80	3000[3]	A tetracycline	South Asia & the Orient
Yersinia pestis	Plague	1-4	Y	up to 100	3000	chloramphenicol tetracycline	Worldwide

Table 12.--Viruses

Causative Agent	Disease	Latent Period (Days)	Vaccine Y/N/D[1]	Mortality (%)	Infectious Dose 50% (ID_{50})	Prophylaxis/ Treatment	Areas of endemicity
CCHFV (Bunyaviridae)	Crimean-Congo hemorrhagic fever	3-6	N	13-40		Ribavirin shown to have experimental effectiveness	Southern former USSR, and Bulgaria; Middle East, Central Asia, Pakistan
CHIK	Chikungunya fever	2-6	D	0-1			sub-Saharan Africa to the Asian tropics
Dengue V. (Flaviviridae)	Dengue Fever	2-7	D	1-20	2	treatment not available; highly incapacitating	Indian subcontinent to Iran & sometimes to the Middle East & Greece, South Pacific, Indochina, Caribbean
Ebola Virus (Filoviridae)	Ebola hemorrhagic fever	2-21	N	50-90		treatment not available	Central Africa, predominantly within 5° of the Equator
EEV (Togaviridae)	Eastern Equine Encephalitis	5-15	Y	50-80		treatment not available	Canada, Eastern U.S., Caribbean states, & South America
HAV (Picornaviridae)	Hepatitis A	10-50	Y				
HPS (Bunyaviridae)	Hantavirus Pulmonary Syndrome (Four Corners Disease	3-10	N	>75		treatment not available	Western US, especially New Mexico, Arizona, Colorado
HTN Hantaan Virus (Bunyaviridae)	Korean hemorrhagic fever	12-33	Y but questionable	1-30		treatment not available	Asia to Central Europe and Scandinavian States

Table 12.--Continued

Causative Agent	Disease	Latent Period (Days)	Vaccine Y/N/D[1]	Mortality (%)	Infectious Dose 50% (ID_{50})	Prophylaxis/ Treatment	Areas of endemicity
Influenza Virus (Orthomyxo-viridae)	The flu	1-3	Y for known/ common strains	0-1		Amantadine (prophy-laxis)	Worldwide; new strains typically originate in China, Southeast Asia
JEV (Flaviviridae)	Japanese encephalitis	5-15	Y	2-50		treatment not available	Far East & Pacific islands
Junin Virus	Argentinean hemorrhagic fever	7-14	D	5-15			
Lassa Virus (Arenaviridae)	Lassa hemorrhagic fever	5-21	D	30-60		Ribavirin shown to have experimental effectiveness	West Africa from Cameroon to Senegal
LCMV (Arenaviridae)	Lymphocytic choriomenin-gitis	6-13	D	low			Worldwide
Machupo Virus (Arenaviridae)	Bolivian hemorrhagic fever	7-14	D	5-15		Antibody treatment effective	Subtropical Argentina and Bolivia
Marburg Virus (Filoviridae)	Marburg disease (hemorrhagic fever)	6-10	N	35			Germany, Yugoslavia & possibly South Africa
RSSEV (Flaviviridae)	Russian spring-summer encephalitis	7-14	Y	0-30		treatment not available	Prevalent in Eastern Europe and Russia; outbreaks peak during June-July

Table 12.--Continued

Causative Agent	Disease	Latent Period (Days)	Vaccine Y/N/D[1]	Mortality (%)	Infectious Dose 50% (ID_{50})	Prophylaxis/ Treatment	Areas of endemicity
RVF (Bunyaviridae)	Rift valley fever	1-5	Y	1-10		Ribavirin & Ribamidine, antibody treatment	Northeastern, Southern, & Eastern Africa
SLEV (Flaviviridae)	St. Louis encephalitis	4-21	N	2-22		none	U.S., Trinidad, Panama
Variola Virus (Poxviridae)	Smallpox	7-16	Y	10-50		Gamma-globulin, thiosemi-carbazone	Worldwide; last known case was Somalia, 1977
VEE (Togaviridae)	Venezuelan equine encephalitis	1-6	Y	0-2	1	no specific treatment necessary	Brazil to Southern U.S. states
WEV (Togaviridae)	Western equine encephalitis	7-21	Y	3-15	low aerosol infectivity		Canada, Western U.S., Argentina, Mexico, Brazil
YFV (Flaviviridae)	Yellow Fever	1-6	Y	5-100		treatment not available	Worldwide, but more common to remote tropical regions

Table 13.--Toxins

Toxin (source)	Type and Effects	Rate of Action	Toxoid Y/N/D[4]	Lethal/ Incapacitating Dose 50% (LD/ID_{50})	Comments
Anatoxin A (very fast death factor) (*Anabaena flos-aquae*)	Lethal, paralytic, bacterial neurotoxin; chemical nerve agent symptoms (seizures and tremors)	5 min		LD_{50} - 170-250 µg/kg	Water soluble; nonpersistent.
Conotoxin (*Conus geographus, C. magus*)	Lethal snail neurotoxin; bleeding at injection site; muscle weakness	5 min		LD_{50} - 3-6 µg/kg	Highly stable
Palytoxin (*Palythoa toxica*)	Most potent non-protein toxin known; lethal coral neurotoxin, muscle paralysis, collapse	5 min		LD_{50} - 0.08-0.4 µg/kg	Soluble in water, pyridine, DMSO & alcohol; persistent
Alpha-latrotoxin	Lethal; spider neurotoxin; paralytic; chemical agent symptoms	5 min to 1 hr		LD_{50} - 10.0 µg/kg	
Batrachotoxin (*Phyllobates aurotaenia*)	Potentially lethal; frog paralytic neurotoxin; may cause bee sting-like pain on contact with broken skin	5 min to 1 hr		LD_{50} - 0.1-2.0 µg/kg	Soluble in organic solvents & alcohol; relatively nonpersistent.
Beta-bungarotoxin (*Bungarus multicinctus*)	Lethal; banded krait snake neurotoxin; paralytic - blocks acetylcholine release.	5 min to 1 hr		LD_{50} - 2.0 µg/kg	Water soluble; relatively nonpersistent.
Cobrotoxin (*Naja naja atra*)	Lethal; Formosa cobra snake neurotoxin; paralytic	5 min to 1 hr		LD_{50} - 75.0 µg/kg	Water soluble; relatively nonpersistent.
Crotoxin (*Crotalus durissus terrificus*)	Lethal rattlesnake neurotoxin; dizziness, sensory and motor depression, collapse, shock	5 min to 1 hr	Y		

Table 13.--Continued

Toxin (source)	Type and Effects	Rate of Action	Toxoid Y/N/D[4]	Lethal/ Incapacitating Dose 50% (LD/ID50)	Comments
Diphtheria toxin (*Corynebacterium diphtheria*)	Lethal bacterial toxin; sore throat; swollen glands	5 min to 1 hr	Y	LD_{50} - 0.03 µg/kg	
Microcystin (*Microcystis aeruginosa*, or *M. cyanea*)	Lethal algal cytotoxin; shivering; stupor	5 min to 1 hr		LD_{50} - 50.0-100.0 µg/kg	Soluble in water & polar organics; Unstable
Notoxin	Lethal; snake neurotoxin; paralytic	5 min to 1 hr		LD_{50} - 20.0 µg/kg	Water soluble; relatively nonpersistent.
Saxitoxin (*Gonyaulax catanella*, or *G. tamarensis*)	Lethal dinoflagellate toxin; numbness; muscle weakness; incoordination; respiratory distress; death due to respiratory paralysis	5 min to 1hr	D	LD_{50} - 10.0 µg/kg	Water soluble; relatively persistent.
Taipoxin	lethal; snake paralytic neurotoxin	5 min to 1 hr		LD_{50} - 2.0 µg/kg	Water soluble; relatively nonpersistent.
Tetrodotoxin or Fugu Poison (*Tetraodontidae* spp.)	Lethal puffer fish toxin; neuromuscular block; numbness; loss of muscle control; voice loss	5 min to 1 hr		LD_{50} - 10.0 µg/kg	Soluble in acidic solution; relatively persistent.
Staphylococcus enterotoxin B (*Staphylococcus aureus*)	Bacterial incapacitant; sudden and often violent onset of vomiting, diarrhea and stomach cramps. Symptoms rarely last beyond a day	0.5 hr to 4 hrs	D	ID_{50} - 0.04 µg/kg	Cimetidine & diatriazem experimentally effective. Resistant to potable quantities of chlorine; water soluble; nonpersistent

Table 13.--Continued

Toxin (source)	Type and Effects	Rate of Action	Toxoid Y/N/D[4]	Lethal/ Incapacitating Dose 50% (LD/ID_{50})	Comments
Abrin (*Abrus precatorius*)	Lethal jequirity plant poison; a cytotoxin with symptoms which are probably similar to ricin			LD_{50} ~ 0.02 mg/kg	Soluble in saline solutions. Moderately persistent. Plant found in Florida and tropical areas.
Aflatoxin B (*Aspergillus flavus*, & *A. parasiticus*)	Potent liver carcinogen; associated with fungal related food poisoning; probably the same symptoms as T-2		N	LD_{50} - 2.0 mg/kg	Exhibits blue fluorescence (related aflatoxins may fluoresce green); soluble in polar organic solvents.
Botulinum (oral) (*Clostridium botulinum*)	Lethal bacterial neurotoxin; drooping eyelids; double vision; dilated pupils; fever; flaccid paralysis; death in 3 - 7 days	1 hr to 12 hrs	Y for types A - E	LD_{50} - 0.00003-0.01 µg/kg	Anti-toxin reduces some side effects. Better to use antibodies for prophylactic treatment. Non-encapsulated toxin persists for about 6-12 hrs; water soluble.
Nivalenol (*Fusarium nivale*)	Lethal fungal cytotoxin; potent hemorrhagic substance; blisters, tissue death, dizziness, nausea, vomiting, diarrhea.	1 hr to 12 hrs		LD_{50} ~ 0.4 µg/kg	Soluble in polar organic solvents.
Ricin (aerosol, skin, oral) (*Ricinus communis*)	Lethal castor bean cytotoxin; nausea; vomiting; cramps	1 hr to 12 hrs	Y	LD_{50} - 3.0 µg/kg	Maintain life support. Water soluble; very persistent

Table 13.--Continued

Toxin (source)	Type and Effects	Rate of Action	Toxoid Y/N/D[4]	Lethal/ Incapacitating Dose 50% (LD/ID_{50})	Comments
Seawasp (box jellyfish) toxin (*Chironex fleckeri*)	Lethal toxin				Research on this toxin has been conducted by Australia
T-2 (skin, aerosol, oral) (*Fusarium tricinctum*)	Incapacitant/lethal fungal cytotoxin; skin reddening, rash, blisters; nausea; bloody vomit; diarrhea	1 hr to 12 hrs	D	ID_{50} - 0.1 mg/kg	Soluble in organic solvents; persistent.
Tetanus (*Clostridium tetani*)	Lethal bacterial neurotoxin; muscle spasms, frequently of the jaw muscles.	1 hr to 12 hrs	Y	LD_{50} - 0.001 µg/kg	

[1]Y indicates that a vaccine is currently available, N indicates that a vaccine is neither in development nor available, and D indicates that work is currently under way to develop a vaccine.
[2]Joseph D. Douglass, Jr., "Who's Holding the Psychotoxins And DNA-Altering Compounds?" Armed Forces Journal International, 130, no. 2 (September 1992), 50-52.
[3]Peter Williams and David Wallace, Unit 731: Japan's Secret Biological Warfare In World War II, (New York, The Free Press, 1989), 198.
[4]Y indicates that a toxiod (which is similar to a vaccine--it confers immunity) is currently available, N indicates that a toxoid is neither in development nor available, and D indicates that work is currently under way to develop a toxoid.

BIBLIOGRAPHY

Books

Alexander, Archibald S., R. R. Baxter, Stewart Blumenfeld, David Brown, Thomas Buergenthal, and Matthew Meselson. The Control Of Chemical And Biological Weapons. New York: Carnegie Endowment For Interantional Peace, 1971.

Atlas, Ronald M. Microbiology: Fundamentals And Applications. 2nd ed. New York. Macmillan Publishing Company, 1988.

Budavari, Susan, Maryadele J. O'Neil, Ann Smith, Patricia E. Heckleman, editors. The Merck Index. 11th ed. Rahway, New Jersey: Merck and Company, Inc., 1989.

Carus, W. Seth. The Genie Unleashed: Iraq's Chemical And Biological Weapons Program. Washington, D.C.: The Washington Institute for Near East Policy, 1989.

Clarke, Robin. The Sileni Weapons. New York: The David McKay Company, Inc., 1968.

Collins, C. H., Patrica M. Lyne, and J. M. Grange. Collins And Lyne's Microbiological Methods. 6th ed. Oxford, England: Butterworth-Heinemann, 1989.

Compton, James A. F. Military Chemical And Biological Agents: Chemical And Toxicological Properties. Caldwell, New Jersey: The Telford Press, 1987.

Cordesman, Anthony H. Weapons Of Mass Destruction In The Middle East. London: Brassey's, a member of Maxwell Macmillan Pergamon Publishing Corporation, 1991.

Douglass, Joseph D. Jr., and Neil C. Livingstone. America The Vulnerable: The Threat Of Chemical/Biological Warfare. Lexington, Massachusetts: Lexington Books, 1987.

Frankel-Conrat, Heinz, Paul C. Kimball, and Jay A. Levy. Virology. 2nd ed. Englewood Cliffs, New Jersey: Prentice Hall, 1988.

Freshney, R. Ian. Culture Of Animal Cells: A Manual Of Basic Technique. 2nd ed. New York: Alan R. Liss, Inc., 1987.

Geissler, Erhard. Biological And Toxin Weapons Today. New York: Oxford University Press, 1986.

Harris, Robert, and Jeremy Paxman. A Higher Form Of Killing: The Secret Story Of Chemical And Biological Warfare. New York: Hill and Wang, 1982.

Harr, Barend ter. The Future Of Biological Weapons. New York: Praeger, 1991.

Hemsley, John. The Soviet Biochemical Threat To NATO. New York: St. Martin's Press, 1987.

Hinds, William C. Aerosol Technology: Properties, Behavior, And Measurement Of Airborne Particles. New York: John Wiley & Sons, 1982.

Kenkel, John. Analytical Chemistry For Technicians. Chelsea, Michigan: Lewis Publishers, Inc., 1988.

le Chene, Evelyn. Chemical And Biological Warfare--Threat Of The Future. Toronto, Canada: The Mackenzie Institute, 1989.

Livinstone, Neil C., and Joseph D. Douglass, Jr. CBW: The Poor Man's Atomic Bomb Cambridge, Massachusetts: Institute For Foreign Policy Analysis, Inc., 1984.

McDermott, Jeanne. The Killing Winds: The Menace Of Biological Warfare. New York: Arbor House, 1987.

Rose, Steven, and David Pavett. CBW: Chemical And Biological Warfare. Boston: Beacon Press, 1968.

Rosebury, Theodor. Experimental Air-Borne Infection. Baltimore: The Williams & Wilkins Company, 1947.

_____. Peace or Pestilence: Biological Warfare And How To Avoid It. New York. Whittlesey House, 1949.

Seagrave, Sterling. Yellow Rain: A Journey Through The Terror Of Chemical Warfare. New York: M. Evans and Company, Inc., 1981.

Silverstein, Robert M., G. Clayton Bassler, and Terence C. Morrill. Spectrometric Identification Of Organic Compounds. 3d ed. New York: John Wiley & Sons, Inc., 1974.

Stockholm International Peace Research Institute (SIPRI). The Problem Of Chemical And Biological Warfare. Vol. 1, The Rise of CB Weapons, by SIPRI. New York: Humanities Press, 1971.

_____. The Problem Of Chemical And Biological Warfare. Vol. 2, CB Weapons Today, by SIPRI. New York: Humanities Press, 1973.

_____. The Problem Of Chemical And Biological Warfare. Vol. 6, Technical Aspects Of Early Warning And Verification, by SIPRI. New York: Humanities Press, 1975.

Williams, Peter, and David Wallace. Unit 731: Japan's Secret Biological Warfare In World War II. New York: The Free Press, A Division Of Macmillan, Inc., 1989.

Government Publications

US. Army. FM 27-10, The Law Of Land Warfare. With change 1. Washington, DC: Headquarters, Department Of The Army, 1956.

US Army. FM 100-5, Operations. Washington, DC: Headquarters, Department Of The Army, 1993.

US Army Medical Research And Development Command (USAMRDC). Final Programmatic Environmental Impact Statement: Biological Defense Research Program. Fort Detrick, Frederick, Maryland: US Army Medical Research And Development Command, 1989.

US Army and US Marine Corps. FM 3-3/FMFM 11-17, Chemical And Biological Contamination Avoidance. Washington, DC: Headquarters, Department Of The Army/Commandant Marine Corps. 1992.

US Army, US Navy, and US Air Force. FM 3-9, Potential Military Chemical/Biological Agents And Compounds. Washinton, DC: Headquarters, Departments of the Army, Navy, and Air Force, 1990.

US Army War College Library. Chemical And Biological Warfare: A Selected Bibliography. Carlisle Barracks, Pennsylvania: US Army War College Library, 1991.

United Nations Group of Consultant Experts on Chemical and Bacteriological (Biological) Weapons. Chemical And Bacteriological (Biological) Weapons And The Effects Of Their Possible Use. New York: United Nations, 1969.

Periodicals

Baker, David. "Chemical And Biological Warfare Agents--A Fresh Approach." Jane's Intelligence Review 5 (January 1993): 42-44.

Barry, John, Daniel Pedersen, and Tom Post. "Planning A Plague?" Newsweek (1 February 1993): 40-41.

Begley, Sharon, John Barry, and Mary Hager. "The Germ Warfare Alert." Newsweek (7 January 1991): 25.

Bermudez, Joseph S. Jr. "North Korea's Chemical And Biological Warfare Arsenal." Jane's Intelligence Review 5 (May 1993): 225-228.

Bradley, David. "Frog Venom Cocktail Yields A One-Handed Painkiller." Science 261 (27 August 1993): 1117.

Buckelew, Alvin H. "Security Without Constraints." Security Management (June 1987): 48-54.

Cordesman, Anthony H. "Creating Weapons Of Mass Destruction." Armed Forces International 126 (February 1989): 54-57.

Douglass, Joseph D. Jr., and H. Richard Lukens. "The Expanding Arena Of Chemical-Biological Warfare." Strategic Review 12 (Fall 1984): 71-80.

Douglass, Joseph D. Jr. "Biochem Warfare: A Warning." Defense Science 2002+ 3 (October 1989): 66-69.

_____. "The Expanding Threat Of Chemical-Biological Warfare: A Case of U.S. Tunnel-Vision." Strategic Review 14 (Fall 1986): 37-46.

_____. "BioChem Warfare: New Dimensions And Implications." Defense & Foreign Affairs. 15 (April 1987): 41-45.

_____. "Soviets Surge In Biochemical Warfare: West Remains Drugged With Apathy." Armed Forces Journal International 126 (August 1988): 54, 58.

_____. "Who's Holding The Psychotoxins And DNA-Altering Compounds?" Armed Forces Journal International 130 (September 1992): 50, 52.

Eisenstadt, Michael. "Syria's Strategic Weapons." Jane's Intelligence Review 5 (April 1993): 168-173.

Gander, Terry J. "Iraq -- Chemical Warfare Potential." Jane's Soviet Intelligence Review 10 (October 1990): 441.

Glickman, Leonard. "Biochemical Warfare Must Be Examined, Controlled." Journal Of Defense & Diplomacy 6 (January 1988): 56-58.

Goldsmith, Marsha F. "Defensive Biological Warfare Researchers Prepare To Counteract 'Natural' Enemies In Battle, At Home." Journal Of American Medical Association 266 (13 November 1991): 2522-2523.

Haase Ewin, J. V. "Chemical And Biological Warfare: The New Frontier." Asian Defence Journal 6 (June 1986): 84-86.

Harris, Elisa D. "Sverdlovsk And Yellow Rain: Two Cases Of Soviet Noncompliance?" International Security 11 (Spring 1987): 41-95.

Hughes, James M., C. J. Peters, Mitchell L. Cohen, and Brian W. J. Mahey. "Hantavirus Pulmonary Syndrome: An Emerging Infectious Disease." Science 262 (5 November 1993): 850-851.

Ivarsson, Ulf. "Tracking Down Poisons." International Defense Review 26 (October, 1993): 823.

Levinson, Macha. "Custom-Made Biological Weapons." International Defense Review 19 (November 1986): 1611-1615.

Marwick, Charles. "Defense Appears To Have Advantage Over Offense Presently In Biological Warfare." Journal Of American Medical Association 265 (13 February 1991): 700.

Marshall, Eliot. "Hantavirus Outbreak Yields To PCR." Science 262 (5 November 1993): 832 - 836.

McGeorge, Harvey J. "Bugs, Gas And Missiles." Defense & Foreign Affairs 18 (May-June 1991): 14-19.

_____. "Iraq's Secret Arsenal." Defense & Foreign Affairs 19 (January-February 1991): 6-9.

_____. "The Growing Trend Toward Chemical And Biological Weapons Capability." Defense & Foreign Affairs 19 (April 1991): 4-7.

Murray, Charles J. "Detector Identifies Biological Warfare Agents." Design News 48 (20 April 1992): 87-88.

Nichol, Stuart T., Christina F. Spiropoulou, Sergey Morzunov, Pierre E. Rollin, Thomas G. Ksiazek, Heinz Feldman, Anthony Sanchez, James Childs, Sherif Zaki, Clarence J Peters. "Genetic Identification Of A Hantavius Associated With An Outbreak Of Acute Respiratory Illness." Science 262 (5 November 1993): 914 - 917.

Roosevelt, Edith Kermit. "Germ War." International Combat Arms 4 (July 1982): 38-42.

Rose, Stephen. "The Coming Explosion Of Silent Weapons." Naval War College Review 42 (Summer 1989): 6-29.

Stone, Richard. "New Cholera Strain Arouses Concern." Science 261 (27 August 1993): 1119.

Welch, Thomas J. "The Growing Global Menace Of Chemical & Biological Warfare." Defense 89 (July/August 1989): 19-27.

White, Terence, and Kathleen White. "Biological Weapons: How Big A Threat?" International Defense Review 23 (August 1990): 843-846.

Wohl, Richard. "Biological Warfare: Advances Breed New Dangers." Defense Science 2002+ 3 (August 1984): 57-60.

_____. "The Forgotten Threat: Biowar." Defense Science 8 (October 1989): 75-77.

Original Manuscripts

Anderson, W. C. III, and J. M. King. Vaccine And Antitoxin Availability For Defense Against Biological Warfare Threat Agents, Fort Sam Houston, Texas: US Army Health Services Command, Health Care Studies Division, Health Care Studies And Clinical Investigation Activity, 1983. DTIC, ADA 137354.

Beebe, J. M., E. L. Dorsey, N. L. Pollock, and E. E. Johns. The Comparative Responses Of *Coxiella burnetii*, *Pasturella tularensis*, *Pasturella pestis*, And *Serratia marcescens* To Artificial Sunlight At Two Humidities. Technical Memorandum 21. Fort Detrick, Maryland: US Army Biological Laboratories Fort Detrick, 1962.

Calder, Kenneth L. Mathematical Models For Dosages And Casualty Coverage Resulting From Single Point And Line Source Releases Of Aerosol Near Ground Level (U). BWL Technical Study 3. Fort Detrick, Maryland: BW Laboratories, Fort Detrick, 1957. DTIC, AD 310361.

Cherwonogrodzky, J. W., and V. L. Di Ninno. Vaccines, Passive Immune Approaches And Treatment Of Biological Agents. Special Publication No. 155. Ralston, Alberta: Defence Research Establishment Suffield, 1992. DTIC, ADA 254600.

Fess, Kenneth E., and Duane E. Williams. Third World Tactical Ballistic Missiles: A Strategy For Defense (U). Carlisle Barracks, Pennsylvania: U.S. Army War College, 1991.

Mack, Andrew. More Arms, Less Stability: Nuclear, Chemical, And Missile Proliferation In The Asia-Pacific. Canberra, Australia: Peace Research Center, Australian National University, 1991. DTIC ADA 254337.

Ursano, R. J. Psychological And Behavioral Responses To A Chemical And Biological Warfare Environment: Final Recommendations. Bethesda, Maryland: Department Of Psychiatry, F. Edward Hebert School Of Medicine, Uniformed Services Of The Health Sciences, 1988. DTIC, ADA 203675.

Wu, Daniel, and Dale W. Sloop. A Package Of Transport And Diffusion Models For Biological And Toxin Agents. CRDEC TR 86034. Aberdeen Proving Ground, Maryland: U.S. Army Armament Munitions Chemical Command, 1986. DTIC ADB 105057.

Unpublished Materials

Aspin, Les, Secretary of Defense, and General Colin Powell. "Department Of Defense Bottom-Up Review." The Pentagon, Washington, DC, 1 September 1993.

Briefing by US Army Chemical School On Current Chemical And Biological Threats (Unclassified). Fort McClellan, Alabama: US Army Chemical School, August, 1993.

Briefing packet for "Tri-Service Technology Workshop On Biodetection Systems. 12 - 13 May 1992" [May 1992]. US Army Chemical Research, Development and Engineering Center, Aberdeen Proving Ground (Edgewood Area), Maryland.

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